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VOLUME 31 NUMBER 9 SEPTEMBER, 1948

AMERICAN JOURNAL OF OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

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Subscription price in United States ten dollars yearly. In Canada and foreign countries twelve dollars. Published monthly by the Ophthalmic Publishing Company. Subscription and Advertising Office: 664 North Michigan Avenue, Chicago 11, Illinois. Entered as second class matter at the post office at Menasha, Wisconsin.

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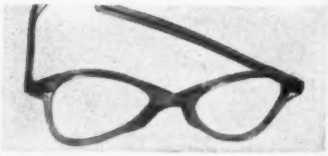
This little girl, so sweet and demure
Is bright for her age, her mother is sure;
In first grade she excells in each letter and song,
Prescribe the "**F-144**" to help her along.

This little fellow, with hair all awry,
Scuffed on the face and patch over eye,
Is not much on study, but rarin' to go;
Action he likes—prescribe "**RODEO.**"

Two solid senders having their day
He's in the groove with the "**MIDWAY**"
Coed — she is well in the know
It's "**HUSSY**" for her in "**CALICO**"

The Queen of the Campus, her manners refined
With a spirit unequalled, all the boys are inclined
To admire her good taste and single perfection
"**WHIMSY**" will be her unquestioned selection.



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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 31 SEPTEMBER, 1948 NUMBER 9

OPTIC NEUROPATHIES: A SIMPLIFIED CLASSIFICATION AND OUTLINE FOR ETIOLOGIC DIAGNOSIS*

THE ESTELLE DOHENY EYE FOUNDATION LECTURE

ALAN C. WOODS, M.D.
Baltimore, Maryland

The confusion in the etiologic diagnosis of diseases of the optic nerve is due to two factors: first, the complexity of the subject and, second, to the fact that the subject lies in several fields—ophthalmology, neurology, and internal medicine. As a result, there has gradually been developed a welter of terms, some highly descriptive, many others highly confusing, all of which have been used both discriminantly and indiscriminantly by various authors. Many of the classifications proposed are so comprehensive and formidable that the average practitioner, confronted with a case of optic-nerve disease, scarcely knows where to begin his investigation. The diagnostic approach to the problem proposed in this paper has at least the advantage of relative simplicity and works out well in actual practice.

GENERAL CLASSIFICATION OF OPTIC-NERVE DISEASE

Primarily, from the viewpoint of etiology, diseases of the optic nerve may be divided into two general classes:

1. Degenerations of the optic nerve secondary to local ocular disease, circulatory failure, local malformations, local accidents or infections, or hereditary in basis.
2. Diseases of the optic nerve due to central nervous system disease, intracranial anomalies, systemic disease, and various intoxications.

GROUP 1

The first group lies predominantly in the field of the ophthalmologist, and requires little comment. A thorough ophthalmic examination by a competent specialist will almost invariably reveal the true nature and cause of the neuropathy. As a rule, little further investigation is required, although in some cases confirmatory evidence may be sought, and in others certain special examinations may be indicated for the exclusion of remote possible etiologic factors.

In this large group are included: (a) atrophies secondary to glaucoma and retinal degeneration (pigmentary degeneration, and so forth); (b) those associated with congenital retinal and often cerebral degeneration (Tay-Sachs's disease, juvenile cerebroretinal degeneration, familial retinal degeneration); (c) atrophies secondary to circulatory failure—embolus of central retinal artery, sclerosis of the nutrient vessels of the optic nerve (atherosclerosis, arteriolar sclerosis, vascular spasm); (d) atrophies secondary to such bony changes as oxycephaly, abnormalities of the optic foramina, fractures of the orbit; (e) neuropathies secondary to orbital conditions—hemorrhage, infections, tumors, and so forth (Leber's disease).

The one essential common denominator of this entire group is that a careful history, a

* From the Wilmer Ophthalmological Institute of The Johns Hopkins University and Hospital. Delivered at Los Angeles, December 18, 1947.

thorough ophthalmic examination including all visual and motor functions, and local or radiographic examination of the orbit will usually reveal the cause of the optic neuropathy. This group of neuropathies therefore offers no general diagnostic problem.

Group 2

The second group of optic diseases includes those associated with central nervous-system disease, intracranial anomalies, systemic disease, and intoxications of one sort or another. When the ophthalmologist is confronted with cases of this type, it becomes his duty: (1) to exclude etiologic factors belonging to the first group; (2) to analyze and to evaluate all the information obtained by the ophthalmic examination and to classify the disease as far as possible; (3) to direct the consultant services of the neurologist, internist, otolaryngologist, and radiographer along the indicated lines; and (4) in the light of all the assembled information, to arrive at the diagnosis and direct the patient to the proper channels for treatment.

Classification of an optic neuropathy

In the classification of an optic neuropathy, the initial step is to decide whether the neuropathy belongs to the primary or secondary group of optic-nerve disease. This requires a word of explanation. It is of course admitted that, in the etiologic sense, there is no such thing as a primary atrophy. The application of the words primary and secondary neuropathies refers only to the ophthalmoscopic examination. Since all diseases of the optic nerve—inflammations, degenerations, and pressure changes—will, if of sufficient severity and duration, ultimately produce either a primary or a secondary atrophy of the nerve, they may first be considered from this viewpoint. "Primary" neuropathy implies that the optic nerve shows no ophthalmoscopic evidence of present or past inflammation or pressure change. "Secondary" neuropathy implies that the optic nerve shows evidences either of present or past inflammation or pressure changes.

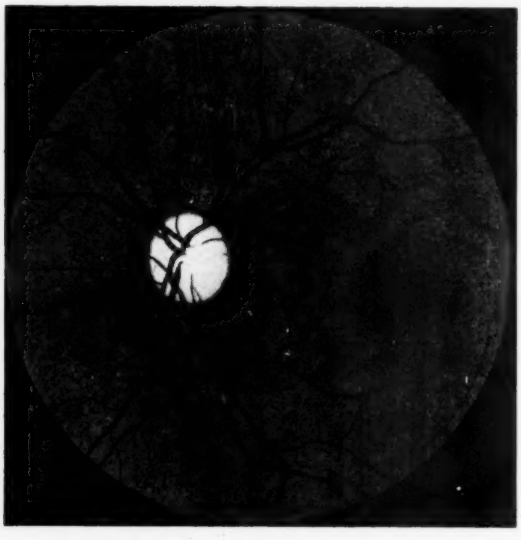

Fig. 1 (Woods). Complete primary optic atrophy.

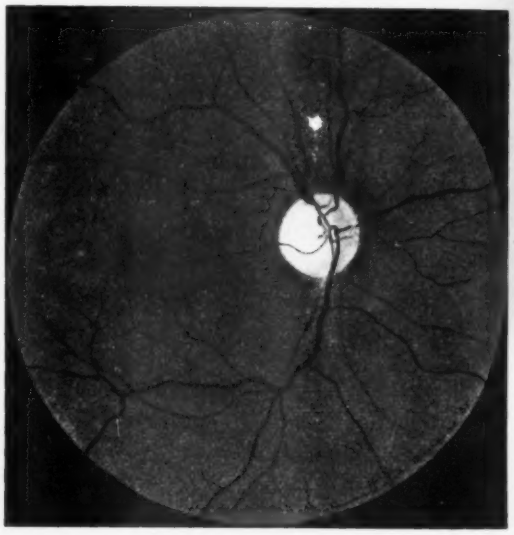

Fig. 2 (Woods). Atrophy of the papillomacular bundle.

Primary neuropathy

A primary optic atrophy may be complete (fig. 1), involving the entire nerve, or may be partial, involving the papillomacular bundle (fig. 2). The abnormality is confined to the optic nerve, the edges are normally outlined, the physiologic cup and lamina cri-

brosa are normal, there is no evidence of edema, congestion, exudation or hemorrhage, or distortion of the neuroretinal outlines. A whitish or bluish-white color, involving either the entire nerve or the temporal sector occupied by the papillomacular bundle, is the one abnormal finding. As the disease progresses, the degree of atrophy increases. Attenuation of the vessels, due to fibrosis in the nerve and up-stream traction, is a late development.

Fig. 3 (Woods). Papilledema.

Secondary neuropathy

The early picture of secondary neuropathy is edema of the nervehead (fig. 3). This may be due either to increased intracranial pressure or to actual inflammation of the nerve. The picture of early pressure changes are edema, actual elevation of the nervehead, blurring of the neuroretinal margins, loss of the physiologic cup from edema, overfilling of the veins, with bending of the vessels at the edge of the edematous papilla (fig. 4).

Hemorrhages and exudates occur later, usually in the peripheral retina, as a result of capillary stasis and local anoxemia (fig. 5). If increased intracranial pressure is established as the cause of the papilledema, the term "choked disc" is used. Actual visible

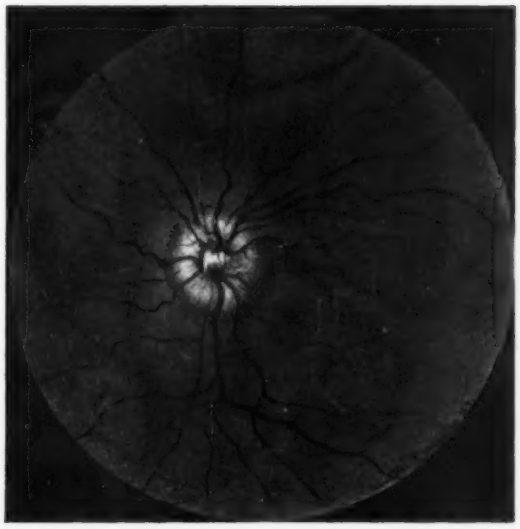

Fig. 4 (Woods). Choked disc—early.

inflammation in the nerve is termed "optic neuritis." In optic neuritis the same edema of the papilla occurs, although often of less degree than in choked disc (fig. 6). There are often other evidences of inflammation, hemorrhages and exudates usually occurring over the nervehead or in the immediately adjacent retina (fig. 7).

The final picture, ophthalmoscopically, is secondary atrophy. The disc is gray, dirty-

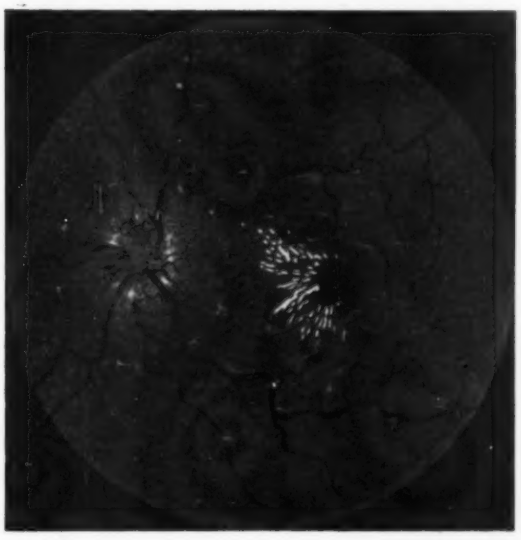

Fig. 5 (Woods). Choked disc—late with beginning secondary atrophy.

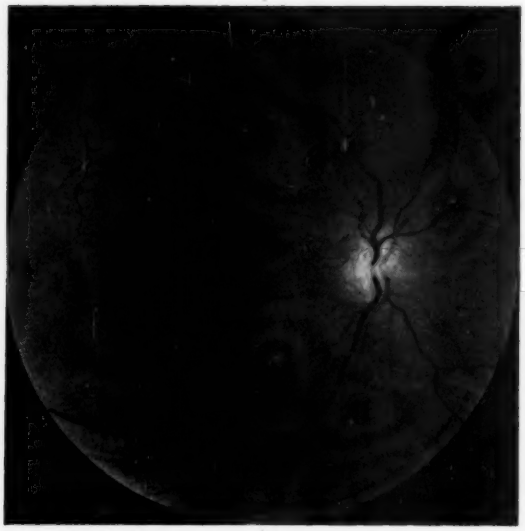

Fig. 6 (Woods). Early optic neuritis.

gray, or reddish-gray in color. The neuroretinal outlines are irregular and slightly distorted with glial tissue. The physiologic cup is lost and the lamina cribrosa is veiled with fibrous or glial tissue which often extends a short distance along the perivascular spaces of the retinal vascular bed as it emerges from the disc and fans out over the retina. This marks the extent of the former pressure or inflammatory changes (fig. 8).

Primary optic atrophy and secondary neuropathies usually arise from distinctly different etiologic factors, although, as will presently be seen, certain conditions may cause either clinical picture, depending on the location of the lesion in the nerve (retrobulbar neuritis and optic neuritis), or on the severity of the intoxication.

Fig. 7 (Woods). Advanced optic neuritis.

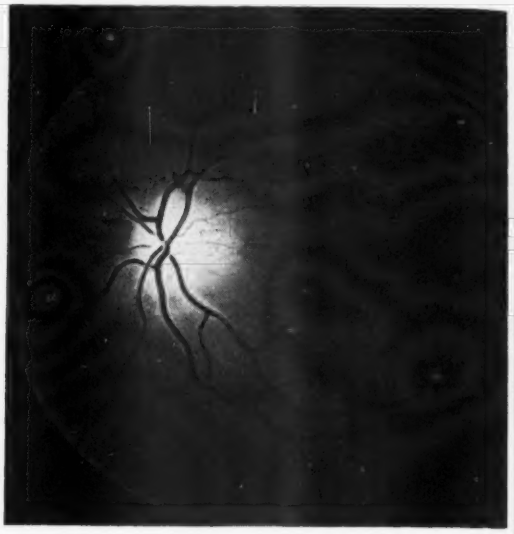

Fig. 8 (Woods). Secondary optic atrophy.

I. Primary Optic Neuropathies

Primary optic atrophy may be total, including the entire nerve in the atrophic process, or partial, including either the peripheral fibers, the papillomacular bundle, or a nerve fiber bundle. The degree of involvement depends in the main on the extent and severity of the underlying lesion, although certain etiologic factors tend to produce total nerve involvement, and other more localized lesions affect especially the papillomacular bundle. The ophthalmologist determines the degree of the nerve involvement by the appearance of the disc, the determination of the visual acuity, and by the visual field changes.

The more important causes producing primary atrophy of the optic nerve may be listed as follows:

1. Central nervous system syphilis: tabes, taboparesis
2. Breaks in the line of conduction: tumors, aneurysms, hemorrhages, trauma

3. Retrobulbar neuritis:
   a. Inflammation in the nerve posterior to the papilla
   b. Demyelinizing disease
   c. Toxic amblyopias

Central nervous system syphilis and large tumors tend ultimately to affect the entire nerve, although in early stages only the peripheral fibers or the papillomacular bundle may be involved. Small tumors and aneurysms often involve a portion of the nerve, the papillomacular bundle, or peripheral sectors. Retrobulbar neuritis usually affects only the papillomacular bundle, although if the local inflammation or the area of demyelinization is large, the entire nerve may be involved. The greater number of the toxins with a selective action of the optic nerve involve the papillomacular bundle only, the pentavalent arsenicals only affecting the peripheral fibers primarily. In massive intoxications the entire nerve may be affected by either group.

Ophthalmic examination does not, as a rule, give sufficient information to differentiate between primary atrophies due to syphilis and those due to breaks in the line of conduction. In general, if the degree of visual failure is slight and the ophthalmoscopic picture of atrophy marked, syphilitic atrophy of the nerve without involvement of the papillomacular bundle is at once suspected. Conversely, if the failure of vision is marked and the picture of atrophy slight, breaks in the line of conduction are suspected. The picture of sharp visual failure, central scotoma, and a normal optic nerve is of course diagnostic of retrobulbar neuritis.

1. Central nervous system syphilis

The primary optic atrophy of syphilis has certain peculiarities. Primarily, in the overwhelming majority of cases, it is associated with a single form of the disease, tabes or taboparesis. It rarely, if ever, occurs in pure paresis. The peculiarities which differentiate it from other forms of syphilis are:

a. Long incubation period
b. The extraordinary selectivity for certain parts of the brain and spinal cord
c. The relative refactoriness to treatment
d. The low reagin content of the blood serum and spinal fluid—the tendency to negative serology
e. The relative paucity or absence of T. pallida in the lesions

The pathogenesis of the primary atrophy of syphilis has long been a problem both to syphilologists and ophthalmologists. Various theories have been proposed. These fall in three general groups.

The first set assumes that the atrophy is due to some form of circulatory failure of the nutrient vessels of the nerve, assumed by some to be related to a disturbance between the retinal and systemic blood pressure. Suffice to say there is no scintilla of evidence in favor of such theories, and there is a wealth of established evidence against them.

The second group of theories attributes the atrophy to adhesions around the chiasm involving the optic nerves—the so-called adhesive chiasmoarachnoiditis. While this condition does occasionally exist, as testified by reputable and trustworthy neurosurgeons, nevertheless it is rarely found in central nervous-system syphilis and when present usually produces a picture midway between primary and secondary atrophy. There is no evidence that it is the cause of the optic atrophy of tabes.

The third group of theories is based on certain experimental evidence and attributes the atrophy to a combination of central nervous-system syphilis and vitamin deficiency, involving both vitamin A and the B-complex. This evidence, which has been fully presented elsewhere, is highly intriguing, fits most of the known facts, and may ultimately be proved to be either the underlying cause or closely related to it. About all we can say at present is that the riddle of syphilitic primary optic atrophy is probably the riddle of tabes itself.

There is much misconception about the

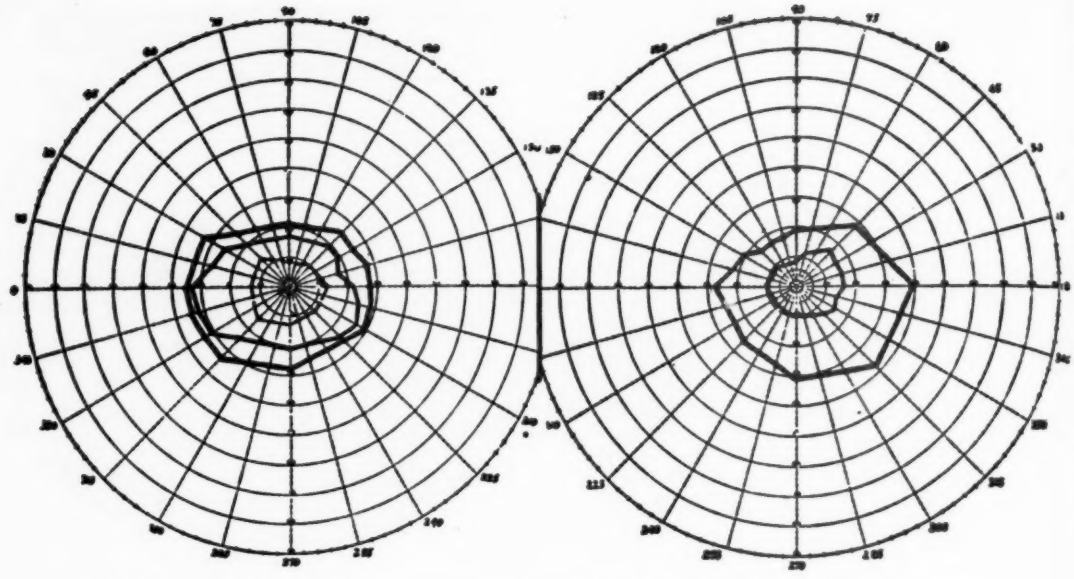

Fig. 9 (Woods). Concentric contraction of visual fields in syphilitic primary optic atrophy.

involvement of the optic nerve in tabes and the resultant visual-field changes. It was formerly supposed that the peripheral fibers were as a rule first involved, and the papillomacular bundle much later, Uthof reporting central scotomas in only two percent of the cases.

This question has been recently investigated and it was found that the visual fields in this group of patients fell into four general classes. These are:

1. Concentric constriction of the field, late loss of vision, occurring in 12.5 percent of the cases (fig. 9).

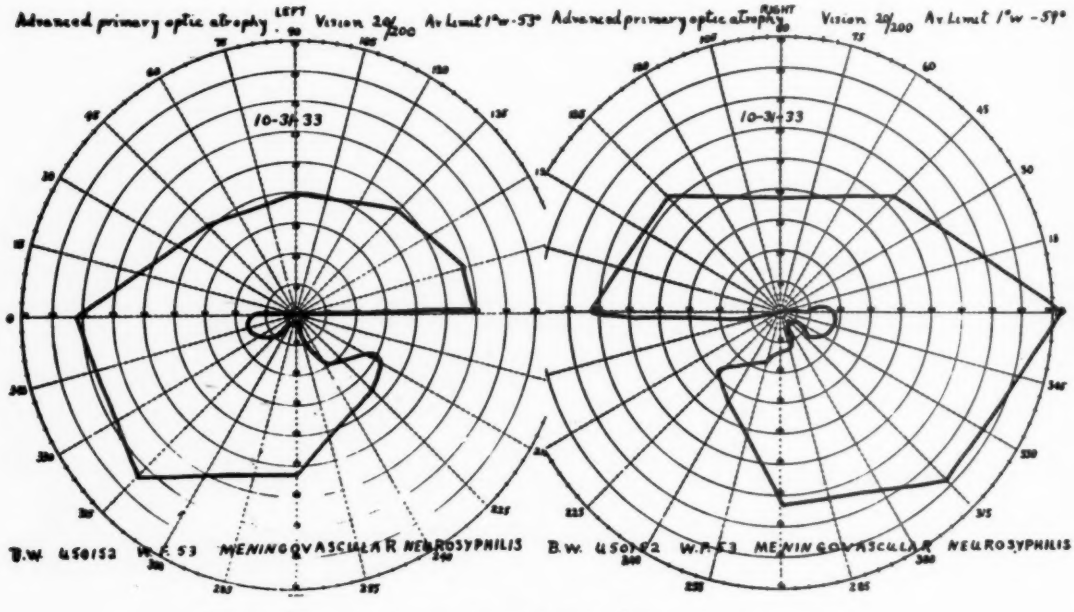
Advanced primary optic atrophy. LEFT Vision 20/200 Av Limit 1°w-53°
10-31-33
B.W. 450152 W.F. 53 MENINGOVASCULAR NEUROSYPHILIS
Advanced primary optic atrophy RIGHT Vision 20/200 Av Limit 1°w-57°
10-31-33
B.W. 450152 W.F. 53 MENINGOVASCULAR NEUROSYPHILIS

Fig. 10 (Woods). Sector defects in syphilitic primary optic atrophy.

2. Sectorlike defects, due to nerve-bundle involvement. The loss of vision in these cases might be early or late, depending on the involvement of the papillomacular bundle. This type of field occurred in 34 percent of the cases (fig. 10).
3. Central, or cecocentral, scotomas, with normal peripheral fields and early loss of vision. This was present in 14 percent of the cases (fig. 11).
4. Central or cecocentral scotomas, associated with changes in the peripheral fields, and early failure of vision, occurring in 40 percent of the cases (fig. 12). Thus, in the series as a whole, 54 percent of the patients had central or cecocentral scotomas with early failure of vision.

Course. The course of syphilitic primary atrophy has been the subject of several studies. Whether the visual failure be early or late, the course is the same. Untreated cases progress to total blindness in from 7 to 9 years, with atrophy of the entire nerve and loss of all light perception (fig. 13).

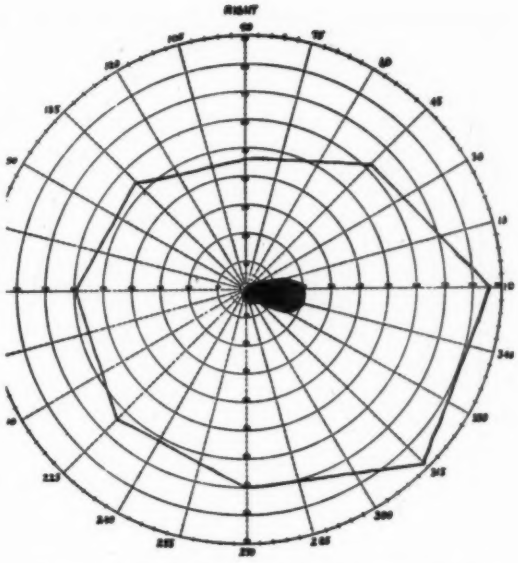

Fig. 11 (Woods). Central and cecocentral scotomas in syphilitic primary optic atrophy.

Treatment. It is traditional that the treatment of syphilitic primary optic atrophy has always been regarded with extreme pessimism by both syphilologists and ophthalmologists. This pessimism is, however, unjustified.

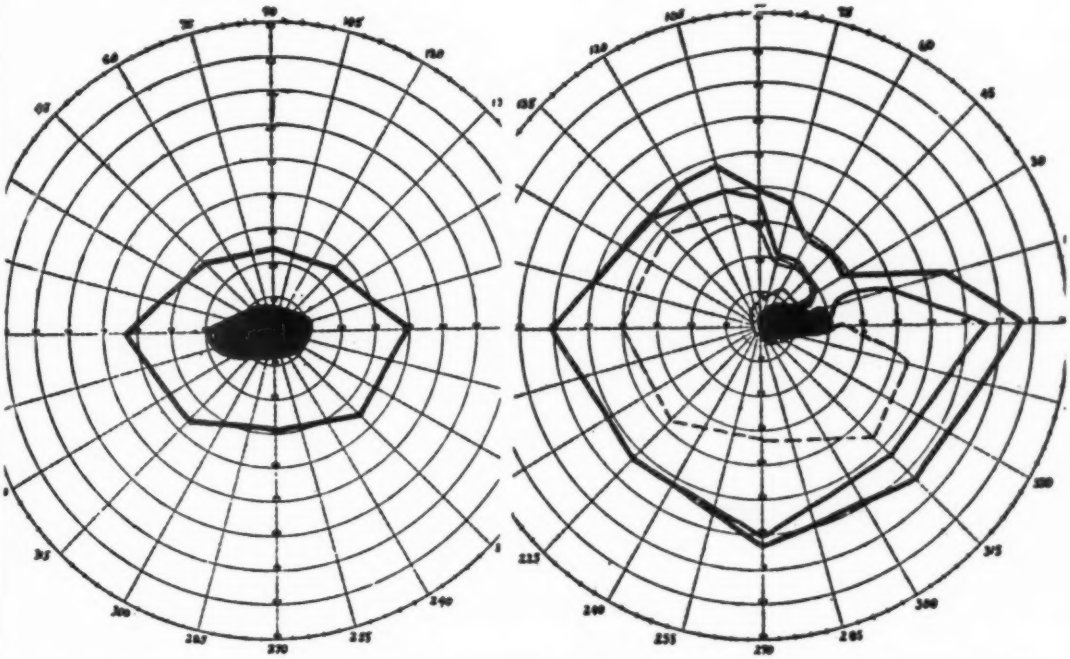

Fig. 12 (Woods). Central scotomas with peripheral field changes in syphilitic primary optic atrophy.

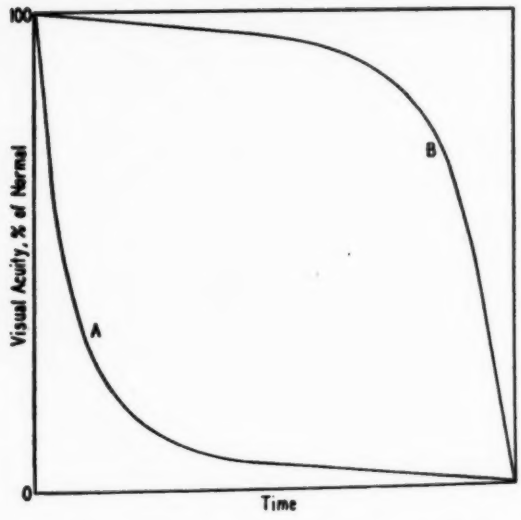

Fig. 13 (Woods). Course of syphilitic primary optic atrophy. Hypothetical curves showing rate of loss of central visual acuity in (A) patients with central and cecocentral scotomas and (B) patients with peripheral constriction of the visual fields only.

No one yet knows what will be the effect of the early treatment of syphilis by penicillin on the later development of primary optic atrophy and tabes. There is some evidence that penicillin is of value in arresting primary optic atrophy, although the results are not uniform. Klauder concludes that since "penicillin does not uniformly benefit the optic atrophy of all patients, the wisdom of its exclusive use in treating the disease is questionable."

The effect of malaria treatment is known. Our earlier experiences with this have already been reported, and our later experiences confirm our first impressions. With this form of treatment only 14 percent of the patients treated lost their vision in the first two years. Thereafter the curve flattened out and 86 percent of the patients retained their remaining vision, of whatever degree it might be (fig. 14).

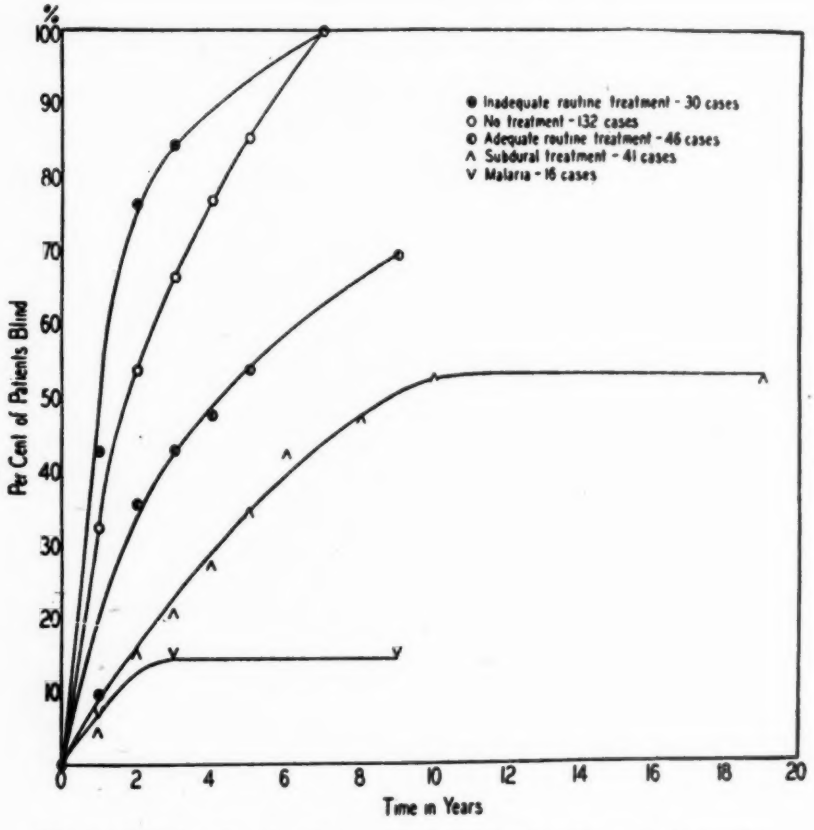

Fig. 14 (Woods). Results of treatment of syphilitic primary optic atrophy.

In his recent book on *Penicillin in Syphilis*, Moore recommends the combination of penicillin and malaria treatment, believing that this form of treatment, capable of completion in about five weeks, may give as satisfactory lasting results as malaria alone followed by prolonged chemotherapy.

2. Breaks in the line of conduction

By atrophies due to breaks in the line of conduction is implied mechanical pressure on the optic nerves either anterior to the chiasm or in the neighborhood of the chiasm, which produce either total or partial primary atrophy of the optic nerves. The most frequent conditions in this category are:

a. Prechiasmal tumors
b. Pituitary tumors
c. Suprasellar tumors (chiasmal syndrome—Cushing's syndrome)
d. Sclerosis of vessels impinging on the chiasm

Prechiasmal tumors. Nothing can be more difficult than the preoperative diagnosis of these tumors. The optic atrophy is usually unequal and of slow development; the visual-field changes are bizarre and unpredictable. Prechiasmal tumors should be suspected only when no other cause for the atrophy can be found and when the field changes are unequal and bizarre. The actual diagnosis can only be made by exploratory craniotomy by the transfrontal route and direct examination of the optic nerves. The usual prechiasmal tumors are meningiomas, dural endotheliomas, and olfactory-groove tumors. They frequently undergo psammoma body changes.

Pituitary tumors. The classic picture of pituitary tumors is destruction of the bony walls of the sella, bilateral primary optic atrophy, bitemporal hemianopia, and radiographic changes in the sella. The older literature occasionally states that the primary optic atrophy secondary to pituitary tumors has a faint yellowish tinge. This is probably incorrect. In appearance the atrophy from pituitary tumors is indistinguishable from

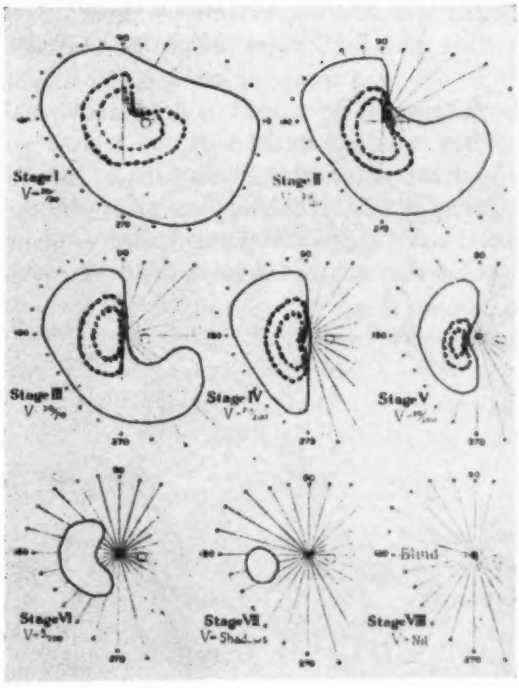

Fig. 15 (Woods). Visual fields in pituitary tumors. (From Cushing and Walker.)

other forms of primary optic atrophy. It should be emphasized that pituitary tumors rarely if ever cause choked disc or papilledema. Hypothetically, this could only occur if the tumor is so placed and of such size that it blocks off the entire intravaginal space.

Despite the volumes which have been written on pituitary tumors, practically nothing has been added to the visual-field studies since the classic paper of Cushing and Walker. These authors divided the perimetric changes into eight grades, from the upper bitemporal slant to total blindness (fig. 15). They emphasized the early loss of the upper temporal color fields, the development of a complete bitemporal hemianopia with involvement of the papillomacular bundle, the gradual limitation of the white fields to the lower nasal quadrants, and the ultimate blindness if the pressure on the nerves was not relieved. Up to and including Stage 4, the condition was reversible, and improvement in the fields and vision might be ex-

pected to follow successful operation.

One additional point should be stressed. In a small percentage of cases, the chiasm is prefixed; that is, instead of lying above and slightly posterior to the sella, the chiasm lies somewhat anteriorly. In such cases an enlarging pituitary might not press on the lower nasal aspects of the optic nerves or on the anterior chiasm but on the posterior portion of the chiasm. Here it might first impinge on the crossed macular fibers and so produce a central scotoma as the cardinal symptom, mimicking the picture of a suprasellar tumor. These cases are rare, but they do occur.

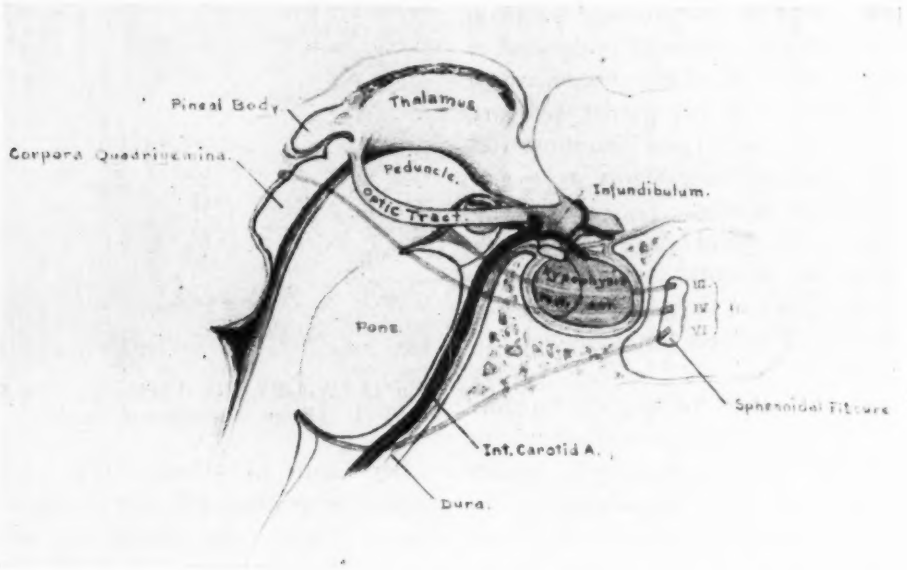

Fig. 16 (Woods). Diagram of relations at chiasm.

Chiasmal syndrome. The chiasmal syndrome is primary atrophy of the optic nerves, or the picture of retrobulbar neuritis, with bitemporal hemianopia or central scotoma and no radiographic evidence of destruction of the sella.

The most frequent tumors causing this syndrome are suprasellar meningiomas, adenomas of the pituitary stalk, craniopharyngiomas (Rathke's pouch tumors), and aneurysms of the anterior portion of the circle of Willis.

The suprasellar meningiomas tend to press on the lower posterior surface of the chiasm, where the crossed nasal fibers lie, thus producing a bitemporal hemianopia.

Adenomas of the pituitary stalk and Rathke's pouch tumors usually press on the posterior lip of the chiasm where they impinge on the crossed macular fibers and so cause a central scotoma. Since there is some intermingling of the lower nasal fibers in the posterior chiasm, they may also produce more or less associated bitemporal hemianopia. Since they all lie centrally, they do not involve any of the nerves going to the extraocular muscles.

Aneurysms of the circle of Willis produce visual-field changes according to their location. The most frequent picture is a central scotoma due to pressure in the papillomacular bundle fibers. Since they lie laterally, they often involve the third cranial nerve.

The actual diagnosis of these tumors is made on the presence of the chiasmal syndrome and exploratory craniotomy. Unfortunately, X-ray examination and ventriculography are of little or no assistance, although at times in the Rathke's pouch tumors the

areas of calcified and keratinized epithelium cast definite shadows in the roentgenograms.

Sclerosis of the vessels, especially the carotids, impinging on the chiasm may produce a primary optic atrophy simulating the so-called "soft glaucoma," with visual-field changes affecting primarily the nasal fields. In his 1916 Bowman lecture, de Schweinitz called attention to the various vascular anomalies which might occur around the circle of Willis, and pointed out how sclerotic vessels might impinge either on the chiasm, the tracts, or the optic nerves and produce bizarre and unusual field changes.

While such changes, binasal hemianopias and bizarre field changes associated with primary optic atrophy, are somewhat rare, in all such cases where no other obvious explanation is present, sclerotic changes in the carotids of the circle of Willis should be considered.

Especially suggestive of pressure changes from sclerosis or aneurysms of the circle of Willis are the altitudinal or unilateral quadratic hemianopias. This is readily understood when one recalls Walker's description of the position of the vessels in relation to the optic neuropathways; the anterior cerebrals passing "over the toes" of the optic nerves, while the posterior communicating pass "under the heels" of the tracts (fig. 16).

The pressure changes from anterior cerebral vessels would produce inferior or altitudinal hemianopia, which might be unilateral if only one nerve is affected. Pressure from the posterior communicating would produce a superior hemianopia, and if only on one side, a quadratic superior defect.

3. Retrobulbar neuritis

Retrobulbar neuritis implies a pathologic process in the optic nerve so far posterior to the papilla that not until a descending atrophy becomes evident at the nervehead is any ophthalmoscopic change evident. Thus in the early stage, Parsons' definition "that neither the patient or the doctor sees anything" is singularly apt.

In general, all the various conditions which cause inflammatory changes at the papilla may likewise cause a retrobulbar neuritis—the essential question is whether the lesion in the nerve is anterior to the lamina so the inflammatory changes are visible, or so well posterior to it that there is no ophthalmoscopic change. However, there are certain conditions which usually produce the clinical picture of retrobulbar neuritis, and others which usually produce the picture of optic neuritis, with visible swelling and inflammation at the nervehead.

The clinical picture of retrobulbar neuritis is as follows:

a. Failure of vision
   1. Acute—sudden loss of central vision
   2. Gradual loss of central vision with fluctuations
b. Ophthalmoscopic picture
   1. Early stages—clear media and normal fundus
   2. Late stages—clear media, atrophy of temporal sector of disc
c. Visual fields
   1. Normal outlines for white and color
   2. Central scotoma varying from relative to absolute white

The more frequent causes of retrobulbar neuritis may be grouped as follows:

1. Intracranial lesions—suprasellar tumors, and so forth
2. Direct involvement of nerve from infections of posterior ethmoids and sphenoids
3. Demyelinizing disease—usually multiple sclerosis
4. Endogenous toxins—diabetes (avitaminosis) and alcohol-tobacco amblyopia
5. Toxic amblyopias

The intracranial lesions, suprasellar tumors, aneurysms of the circle of Willis have already been discussed.

Posterior nasal infection. Retrobulbar neuritis from infection of the posterior nasal sinuses was formerly a fashionable diagnosis. At present it has fallen into disrepute,

and its clinical entity is seriously questioned. While giving full credence to the various clinical studies which have thus cast doubt on it as an actual clinical entity, nevertheless there do appear to be occasional cases of retrobulbar neuritis secondary to infection of the posterior nasal sinuses. Quite probably the changes in the optic nerve are pressure changes only rather than actual infection. Certainly, however, clinical experience indicates that posterior nasal infection must be considered in the search for the etiologic factor in retrobulbar neuritis.

Demyelinizing disease presents a unique problem. The various recognized demyelinizing processes: disseminated sclerosis, acute disseminated encephalomyelitis, neuromyelitis optica, and Schilder's disease of diffuse periaxial encephalitis, may all affect the optic nerve, producing either a retrobulbar neuritis or an actual optic neuritis, according to the location of the lesion. Nevertheless, disseminated or multiple sclerosis usually causes the clinical picture of retrobulbar neuritis while the other demyelinizing diseases tend to give a visible optic neuritis.

It is well recognized that the early retrobulbar neuritis of multiple sclerosis may precede by years the other characteristic changes of the disease, the loss of the abdominal and cremasteric reflexes, the scanning speech, the nystagmus, the ataxia, and other evidences of central nervous-system involvement.

Ophthalmologists are therefore confronted with a group of cases of early retrobulbar neuritis, the attacks beginning with periodic blurring of central vision, the only positive finding being a fluctuating, gradually intensifying central scotoma. There are long remissions between the attacks, which gradually become more severe.

No other etiologic factor can be discovered or suspected; the early neurologic examination is negative; laboratory tests show nothing. Only after several years do the development of the other neurologic manifestations of the disease cement the diagnosis. Few cases can be more exasperating, and all too frequently these patients are subjected to exploratory craniotomies and drainage of the posterior nasal sinuses. No blame can be directed to the ophthalmologist, the neurologist, the neurosurgeon, or the otolaryngologist for such mistakes. The intrinsic baffling nature of the disease is responsible, and until some more accurate method of early diagnosis is developed, such mistakes will continue to be made in the best clinics.

Endogenous toxins. The retrobulbar neuritis from endogenous toxins is interesting. For a long time tobacco-alcohol amblyopia has been recognized as a clinical entity, with the peculiar dumb-bell scotomas involving the blindspot. It has also been recognized that individuals with some debilitating disease, especially diabetes, are especially prone to this type of amblyopia. Only in recent years has it been recognized that the underlying cause is an avitaminosis involving the B-complex, especially thiamine chloride. Fortunately, a form of therapy other than total abstinence is thus afforded sufferers with this unfortunate condition.

The toxic amblyopias affect vision in difference ways. The majority of the known poisons which act on the optic nerve affect primarily the papillomacular bundle. Chief of these are methyl alcohol, carbon disulphide, lead, iodine, thallium, aspidium, salicylates, and aniline dyes.

Methyl alcohol in the commercial form is a universal poison. The others usually produce symptoms only in certain individuals, after varying degrees of exposure.

Quinine amblyopia, on the other hand, results from degeneration of the ganglion cells of the fovea, in individuals with an idiosyncrasy, with resultant ascending atrophy of the papillomacular bundle.

Most important of these especially selective drugs are the pentavalent arsenicals, especially tryparsamide. These arsenicals act first on the peripheral fibers of the optic nerve sparing the papillomacular bundle until the last stages of intoxication. The visual

fields are characteristic with marked constriction above and below (fig. 17). Again, only a small percentage of the population appear susceptible to the toxic action of tryparsamide, and in these, if the field changes are detected early and the drug stopped, central vision can be saved. If, however, the drug is continued and the early visual symptoms ignored, total blindness inevitably results.

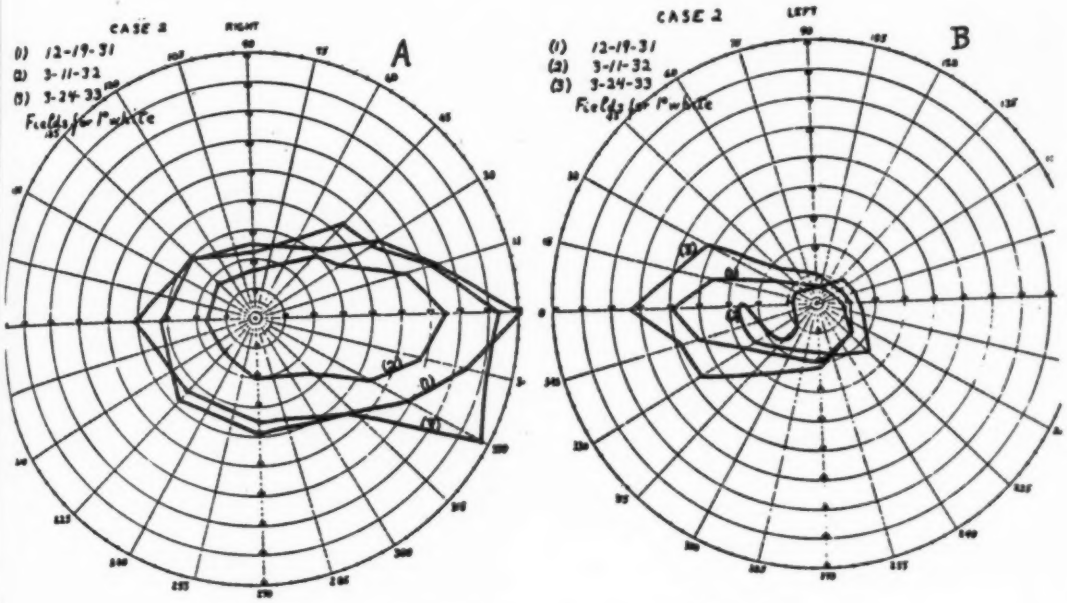

Fig. 17 (Woods). Visual fields in tryparsamide poisoning.

In any event, the usual end-picture of retrobulbar neuritis, unless the cause is early determined and removed when possible, is the same—temporal pallor of the optic nerve from papillomacular-bundle atrophy, with an absolute central scotoma and greatly reduced central vision.

II. Secondary Optic Neuropathies

Optic neuritis and choked disc—two distinct conditions—both produce the same end-picture of secondary optic atrophy. If the patient is encountered in the early stages of papilledema, every effort must be made to differentiate between optic neuritis and choked disc. If the patient is encountered in the final stage of secondary atrophy, the possibility of either primary condition must be considered.

In general, in optic neuritis the inflammatory symptoms are more marked; there is a greater tendency to hemorrhages and exudates over the disc; there is a greater tendency for the inflammatory process to extend out over the immediately adjacent retina; the actual elevation of the disc is usually moderate, usually two diopters or less; and the visual loss may be either slight, if only the peripheral fibers are involved, or severe if the papillomacular bundle is involved.

In the early stages of choked disc which is, by definition, the ophthalmoscopic evidence of increased intracranial pressure, the edema is first manifested at the upper and lower poles of the disc, where the fibers supplying the upper and lower retina are crowded together (fig. 18). In the latter stages, the elevation of the disc becomes high, the physiologic cup is lost, the veins become engorged, edema vesicles form beneath the internal limiting membrane of the retina in the macular area (fig. 19), and ultimately there is a

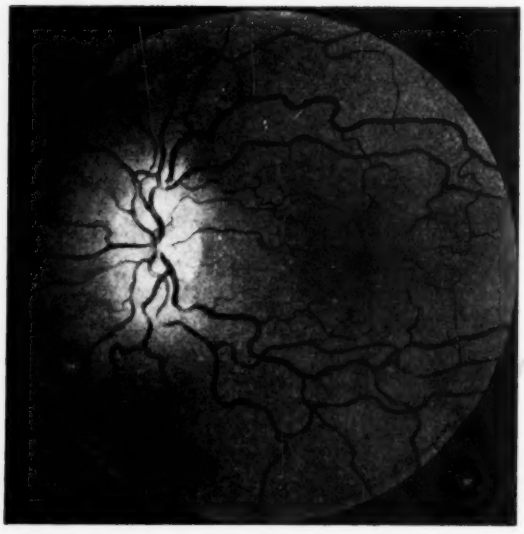

Fig. 18 (Woods). Early choked disc.

full macular figure, with areas of degenerated nerve fibers and hemorrhage, the result of prolonged anoxemia and capillary stasis (fig. 20). While the edema alone may spread out a short distance into the adjacent retina, the maximum elevation is over the papilla itself. The actual elevation of the disc is usually high, often up to 4 to 6 diopters. The visual loss in the early stages, even with high degrees of swelling, may be minimal. Indeed, it is amazing how long the optic-nerve fibers can stand high degrees of pressure from edema alone without atrophy.

These are the general criteria which guide the ophthalmologist in the differentiation of optic neuritis and choked disc. Admittedly, in many cases, the differentiation is presumptive, and in other cases, cannot be made at all. When this is the situation, the etiologic study must proceed upon both presumptions until the actual underlying cause of the papilledema is established.

Optic neuritis is commonly found in the following conditions, excluding such orbital conditions as infections, periostitis, and so forth:

1. Intracranial infections
2. Acute febrile disease
3. Severe toxic amblyopias—especially methyl alcohol
4. Early syphilis
5. Demyelinizing disease—especially neuromyelitis optica
6. Tuberculosis of the sheaths of the optic nerve—(a hematogenous infection or extension of a tuberculous meningitis)

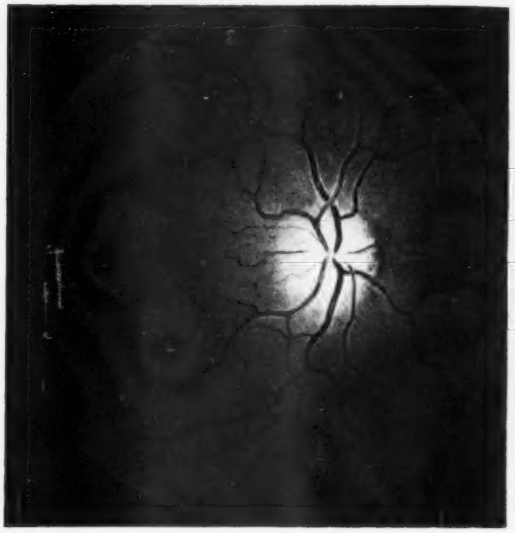

Fig. 20 (Woods). Advanced choked disc.

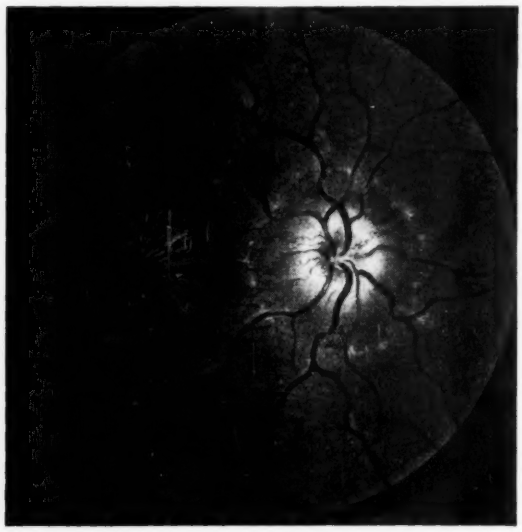

Fig. 19 (Woods). Moderately advanced choked disc.

Few of the conditions require special comment. The optic neuritis complicating a meningitis is obviously the result of direct

extension of the inflammation to the sheaths of the nerve. The optic neuritis of acute febrile disease is an expression of a general toxemia. In severe methyl-alcohol poisoning, with a manifest optic neuritis, the symptoms of systemic intoxication are so marked there is no doubt of the diagnosis. The most interesting conditions in this category are the optic-nerve inflammations of early syphilis, neuromyelitis optica, and tuberculosis.

Early syphilis. The incidence of actual optic neuritis in early syphilis is unknown. The Coöperative Clinical Group reported optic-nerve changes in 11 percent of 3,244 patients with early syphilis, the changes varying from simple hyperemia of the nerve up to an outspoken neuritis.

The one amazing thing about the optic neuritis of early syphilis is how intense the optic neuritis may be without provoking any marked visual symptoms. For this reason it is often detected only as a result of a thorough physical examination. If only the peripheral fibers are involved, as is often the case, there may be no visual changes other than a slight peripheral constriction of the field. If the papillomacular bundle is involved, which is frequent, the visual failure will of course be marked.

Irrespective of the degree of early visual failure, the general rule is that the patients recover from the optic neuritis of early syphilis with little or no loss of vision; some irregularity of the neuroretinal outlines and gliosis over the cup being the only ophthalmoscopic residua. Only in extremely severe cases does secondary optic atrophy and serious impairment of vision result.

Neuromyelitis optica presents a distinct problem, much like that of multiple sclerosis. Indeed the similarity is such that neuromyelitis optica is considered by some to be an acute disseminated sclerosis, although various clinical and histologic differences have been pointed out (Walsh).

Essentially, the disease is a spotty demyelinizing disease with scattered lesions throughout the gray matter especially, with a marked tendency to affect the optic nerves and the spinal cord. In the classic form, the picture is optic neuritis with loss of vision, and a preceding, concomitant, or following paraplegia. In the early stages, there is orbital pain and in the cases with paraplegia often severe root pain. At the onset there is usually a mild fever and pleocytosis of the spinal fluid. The tendency is to recovery both of vision and limb function, although some patients are permanently blind, and some die.

Thus, in the classic form of the disease there is no diagnostic problem. However, there are an appreciable number of cases that show only the eye signs, orbital pain, especially on movement of the eyes, followed by the development of low-grade papilledema without marked evidences of hemorrhage or exudation, loss of vision, usually a central scotoma and sector defects from nerve-bundle involvement, and only minimal evidences of spinal irritation or associated systemic reaction. The disease may be unilateral or bilateral. The most profound investigation reveals no other cause for the optic neuritis.

Gradually, over a period of weeks, the patients slowly recover, without the later development of the expected paraplegia. How these cases are classified—as acute disseminated sclerosis or neuromyelitis optica or by some other name—is an academic question. The essential thing is to realize these cases are a definite clinical entity and recurrences may follow, even years after apparent total recovery.

Tuberculosis is a rare cause of optic neuritis. When it does occur, it is the result either of an extension of the tuberculosis process along the sheaths of the nerve, and into the nerve itself along the pial septa, or the result of a tuberculous periphlebitis of the central retinal vein. In either case the process is destructive. Secondary glaucoma usually results from thrombosis of the posterior veins, and destruction of the optic nerve with secondary atrophy follows. Fortunately the condition is rare and the patients usually die from the concurrent infection.

Comment. These are the main causes of optic neuritis. It may also occur in a variety of rarer conditions, some fungus infections, encephalitis of various causes, Friedreich's ataxia, systemic allergic reactions, Pick's disease, and so forth. These are, however, rare occurrences, and usually present no diagnostic problem.

If optic neuritis is excluded by ophthalmic and clinical examination, the diagnosis of the papilledema as a choked disc is justified. Choked disc must be regarded as the ophthalmoscopic evidence of increased intracranial pressure, and the clinical investigation of the patient extended to establish and determine the course of such pressure.

Examination. The immediate examinations are full perimetric studies and examination of the extraocular muscles to determine any present or latent paralyses. A complete neurologic examination is then indicated to detect any associated neurologic changes. This should be followed by lateral stereoroentgenograms of the skull to detect any pressure changes or abnormal shadows. Lastly comes the serologic examination, cell count, and manometric pressure reading of the spinal fluid. The one caution to be remembered in relation to the spinal puncture is that, in high degrees of choked disc, care should be exercised in the release of spinal fluid pressure, on account of the danger of damage to the brain stem at the foramen magnum.

Diagnosis. On the basis of this ophthalmic and neurologic information, the diagnosis can usually be made between intracranial tumor, which accounts for approximately 75 percent of all choked discs; central nervous system syphilis, which accounts for probably 10 to 15 percent of the cases; and the various other rarer causes (pseudotumor, brain abscess, hydrocephalus, subdural hemorrhage, meningitis, and so forth), which account for the remaining 10 or 15 percent of the cases.

If brain tumor is the underlying cause, a knowledge of the various syndromes caused by tumors at various intracranial levels may greatly help to localize the lesions. These various syndromes have been outlined elsewhere.* They are the pituitary syndrome, the chiasmal syndrome, the Foster-Kennedy syndrome for frontal-lobe tumors, the indefinite changes produced by parietal-lobe tumors, the often more precise changes which accompany temporal-lobe tumors, the characteristic pineal-gland syndrome, the well-known occipital and angle syndromes, and the exact changes produced by tumors at various levels of the brain stem—the so-called Benedikt's, Weber's, Nothnagel's, de Raymond's, Millard-Gubler, and Foville's syndromes. However, only in a limited number of cases, especially those of the chiasmal area, can an exact location be made by the ophthalmic, neurologic, and radiographic examinations. Ventriculography is, of course, the final resource in cerebral localization.

There remains one perplexing class of cases to mention briefly. These are the cases of pseudopapilledema and pseudoneuritis.

Pseudopapilledema occurs usually in hyperopic individuals. The disc margins are blurred, the physiologic cup indistinct, and at times the veins may appear overfilled. The absence of symptoms and the usual presence of hyperopia should put the ophthalmologist on guard. Examination with the binocular ophthalmoscope reveals there is usually no actual elevation of the papilla. There are, however, a few such cases in which the disc may be slightly elevated and still the most exhaustive investigation reveals no organic lesion, the ophthalmoloscopic picture remains unchanged over years of observation, and the patients develop no symptoms. The condition has been attributed to narrowing of the porus opticus.

Pseudoneuritis optica is a somewhat similar picture, but in addition there is usually glial tissue over the disc, and it may resemble a secondary atrophy in color. The condition is due to excessive formation or

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* Woods, A. C.: Ocular manifestations of intracranial tumors. South. Surgeon, **5**:91-114, 1936.

defective atrophy of the tissue which formed Bergmeister's papilla and the hyaloid system. A careful ophthalmoscopic examination usually raises suspicion of the true nature of both of these conditions, but even to the most meticulous ophthalmologist, they remain a source of worry. Fortunately both conditions are comparatively rare.

Conclusion

In this discussion I have endeavored to present a simplified approach to the etiologic diagnosis of optic neuropathies, to give a general classification of the more important causes, and to comment briefly on some of the more interesting points. There remains the question of how such cases should be handled by the ophthalmologist in actual practice. Here I think the answer is fairly obvious.

First, the ophthalmologist should have a clear concept of the various conditions which produce changes in the optic nerves.

Second, his ophthalmic examination should be precise and exhaustive and should enable him, in the majority of instances, to place the individual case in one or the other of the two large groups I have outlined early in this paper, and to classify it as a primary or secondary neuropathy.

Third, if the case falls into the second group, or if there is any question latent in his mind, he should outline the investigation to be followed. This investigation should routinely include a neurologic examination, a general physical examination with serologic and routine examination of the blood and spinal fluid, a lateral stereo of the head, and a clinical and radiographic examination of the posterior nasal sinuses.

Fourth, the ophthalmologist should remain captain of the team until the indications are definite where the patient should be referred for treatment or further investigation.

It is amazing that in the overwhelming majority of cases the information derived from the above examinations permits an immediate and exact diagnosis and establishes the underlying cause of the optic neuropathy.

To my mind the most important point in the etiologic diagnosis of optic neuropathies is that the ophthalmologist should not evade his responsibility. To content himself with the diagnosis of an optic neuropathy secondary to some systemic or intracranial disease and thereupon turn the case over *in toto* to a consultant is an evasion of duty. The neurologist, internist, and neurosurgeon are of necessity and training largely ignorant of the many and diverse causes of optic-nerve disease. While each consultant may be preëminent in his own field, the basic problem is an ophthalmic one; the investigation should be directed by the ophthalmologist; the diagnostic survey assembled by him and correlated with the ophthalmic examination. I believe it is only by this means that the correct diagnosis will be reached in the greatest number of cases.

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THE USE OF BACITRACIN IN OCULAR INFECTIONS*

PART I. TOLERANCE AND PERMEABILITY IN THE RABBIT EYE

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Following the successful application of penicillin and streptomycin in ocular infections, the reported results following the use of a relatively new antibiotic, bacitracin, in various surgical infections stimulated our interest as to its applicability to infections of the eye. Meleney and his coworkers[1, 2] have shown that in general bacitracin is effective against the same bacteria as penicillin and, in addition, report that, where a difference in susceptibility exists, the organisms are more often susceptible to bacitracin than to penicillin in a ratio of 5 to 1. Furthermore, there is no evidence that bacteria can inactivate bacitracin, as is the case with penicillin (penicillinase).

These reports seemed of sufficient importance to warrant undertaking the studies reported here to ascertain the applicability of this antibiotic to ocular infections.

A. THE EFFECT OF BACITRACIN ON THE CORNEAL EPITHELIUM

Saline solutions containing 1,000 and 5,000 units bacitracin per ml. were applied to a series of 12 normal rabbit eyes as drops at one-half hour intervals for six doses, and also in 6 other animals as a constant corneal bath for periods up to one hour. In no case was there any gross evidence of irritation. Upon staining the corneas with fluorescein, no staining areas were visible.

When bacitracin was applied to the corneas for periods up to 30 minutes as a dry powder, then washed out with water, no gross evidence of irritation was apparent. However, upon staining with fluorescein, micropunctate areas became visible. These represented but a transitory injury since, in 24 hours, the eyes were normal in all appearances, and the corneas failed to give a staining reaction.

Another method employed in the study of local toxicity is to observe the effect of the drug upon the regeneration of the corneal epithelium. In a series of 15 rabbits, 5-percent cocaine was applied to both eyes to anesthetize and to facilitate the denudation of the epithelium. The epithelium was removed by rubbing with sterile dry gauze and checked for completeness of abrasion by staining with fluorescein.

Five rabbits received in one eye, 2 drops of the saline solution containing 5,000 units bacitracin per ml., 3 times daily, while the other eye serving as a control received a similar amount of saline. The control eyes healed completely without scarring in 3 to 4 days; whereas, only one receiving the bacitracin healed by the 4th day, and the other 4 remained unhealed when observed on the 9th day. Eventual healing occurred with vascularization and opacities of the corneas. Eyes similarly treated with the solution containing 1,000 units bacitracin per ml. showed no difference in healing rate from that of the saline controls.

Since the rabbit cornea is more sensitive to irritating substances than the human cornea, it is evident that bacitracin in saline solution of a concentration of 1,000 units per ml. may safely be applied to the surface of the human eyeball under any condition. However, if the corneal epithelium is intact,

* Read before the Ophthalmic Section, New York Academy of Medicine, November, 1947. This research was done under a contract between the Office of Naval Research and Northwestern University.

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bacitracin in any concentration, or even as the dry powder, is well tolerated.

B. Permeability of the cornea to bacitracin

For these studies, penetration was observed in a group of 24 albino rabbits under various conditions: (a) where the cornea remained intact, (b) was traumatized, or (c) was inflamed as a result of induced infection.

Rabbits were first anesthetized by the intravenous injection of nembutal. The bacitracin solution was applied as a constant corneal bath by means of a plastic eyecup for periods ranging from 4 to 60 minutes. At the end of the respective periods, the excess of bacitracin was removed from the surface of the eyeball and the adnexa by thoroughly washing with a stream of normal saline. The aqueous was then withdrawn through a limbal puncture by means of a sterile 27-gauge needle. The bacitracin content was determined by the serial dilution method, using the Chanin-Strain Streptococcus hemolyticus, kindly furnished by Dr. Frank L. Meleney. The results of these experiments are shown in Table 1.

TABLE 1

Penetration of bacitracin (1,000 units per ml. saline) through the cornea

Condition of Cornea	No. of Eyes	Duration of Therapy Corneal Bath (Minutes)	Bacitracin Units* per ml. of Aqueous	Remarks
Intact	3	30	0, 0, 0	Non-irritating
Abraded (Amt.)				
100%	2	60	40, 40	Healed within 4 days
100%	2	30	20, 20	Healed within 4 days
50%	2	30	20, 20	Healed within 4 days
25%	3	4	0, 0, 0	Healed within 4 days
25%	2	30	4, 8	Healed within 4 days
Stippled	2	15	0, 0	Healed within 4 days
Stippled	4	30	0, 0.66, 1.0, 2.0	Healed within 4 days
Inflamed (Vaccinia virus)	8	30	2–16 (Av. 6.6)	
Ion transfer				
Intact Cornea	2	15	0, 0	Punctate stippling
Intact Cornea	4	30	0, 0, 0.5, 1	Corneal edema
25% Abrasion	3	4	0, 0, 0	
25% Abrasion	2	30	4, 8	Corneal edema
Aerosol O. T. (1 Drop in 1.0 ml. Bacitracin)				
0.5%	1	60	8	
0.5	1	5	0.12	Staining of cornea, chemosis, and corneal edema
0.1	2	30	4, 4	
0.05	2	30	2, 4	
0.01	2	30	0.5, 1	

* Hemolytic serial dilution method, using the Chanin-strain Streptococcus hemolyticus, kindly furnished by Dr. Frank L. Meleney

From the data presented it will be seen that bacitracin does not penetrate the normal cornea in a period of 30 minutes when applied in a concentration of 1,000 units per ml. In our previous studies on streptomycin,[3, 4] it was shown that, although penetration does not occur in the intact cornea, it readily penetrates following corneal damage.

Similar studies were therefore conducted using bacitracin. It was found that bacitra-

cin behaves in a similar manner. As shown in Table 1, the amount of penetration varies somewhat but not in an absolute manner with the degree of denudation and period of application.

In studies with the inflamed cornea, resulting from the application of the vaccinia virus, penetration in two cases with a 30-minute application of the corneal bath was similar in amount to that occurring in a 25-percent abrasion of the cornea. It is of interest to note that the infected lesion produced by the vaccinia covered about 25 percent of the corneal area.

As observed from Table 1, no detectable amount of bacitracin was found in the aqueous of an eye with intact cornea subjected to ion-transfer (iontophoresis) therapy for a period of 15 minutes. Small amounts of the antibiotic were present after 30 minutes. Application of fluorescein to the cornea following ion-transfer of 15 to 30 minutes in nearly all cases showed punctate staining.

The amount of bacitracin found in the aqueous after ion-transfer was no greater than after a corneal bath containing the same concentration of bacitracin applied for the same time to a cornea which had previously received a similar amount of damage by mechanical means. (Compare amounts for stippled cornea and ion-transfer intact cornea 15 and 30 minutes.)

It was also shown that the passage of bacitracin was no greater with 25-percent abrasion plus ion-transfer than with 25-percent abrasion plus corneal bath alone. From the above data, we conclude that the passage of bacitracin through the cornea does not depend upon ion-transfer per se so much as upon associated epithelial damage. Furthermore, since increased permeability was observed to occur with lesions produced by various means—scraping, a surface-active chemical agent, vaccinia, and during ion-transfer (iontophoresis)—we are led to conclude that the mechanism effecting an increase in penetration through a damaged cornea is essentially the same irrespective of the agent employed.

C. Effect of intraocular injection of bacitracin

Since at present bacitracin cannot be administered parenterally because of its toxicity and, as already shown, it does not penetrate the normal cornea, it was decided to study the effect of direct injection into the vitreous.

For this purpose, 0.1 ml. of a saline solution containing 100 units bacitracin was injected by means of a 27-gauge needle in the region of the equator of the eyeball, taking care to deposit the bacitracin as near the center of the vitreous as possible. The eyes were observed daily with the ophthalmoscope and with the slitlamp.

Minimal to moderate degrees of vitreous opacities were observed immediately following the injection. Usually these disappeared over the course of several weeks. In only 2 of 8 eyes were opacities observed at the termination of the experiment one month later.

At this time two small linear vitreous opacities were observed in one eye, and several fine vitreous opacities in the second. Since no histologic sections were made of these eyes, minute lesions may have escaped observation.

Lens opacities in these early experiments were present in four eyes and were attributed to injury occurring as a result of contact with the needle. In a later series of infected eyes treated by similar injection of bacitracin, rarely was a lens opacity observed. On the basis of size, it is postulated that injection of bacitracin into the human eye should be well tolerated.

In the second part of this report which will be published next month the effect of bacitracin therapy in experimental and clinical ocular infections will be considered.

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References

1. Johnson, B. A., Anker, H., and Meleney, F. L.: Bacitracin: A new antibiotic produced by a member of the B. subtilis group. Science, **102**:376, 1945.
2. Meleney, F. L., and Johnson, B.: Bacitracin therapy: The first hundred cases of surgical infections. J.A.M.A., **133**:675, 1947.
3. Bellows, J. G., Burkholder, M. M., and Farmer, C. J.: Streptomycin in experimental ocular infections. Proc. Soc. Exper. Biol. & Med., **65**:17, 1947.
4. Bellows, J. G., and Farmer, C. J.: Streptomycin in ophthalmology. Am. J. Ophth., **30**:1215, 1947.

SURGERY OF CONGENITAL CATARACTS*

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The advent of the rubella cataract has served to stimulate a new interest in the surgical treatment of congenital cataracts. During the past 25 years only a few scattered papers on the subject have appeared in the literature. Of the more recent ones, that of Falls[1] is the most comprehensive. In the present paper no attempt will be made to present a statistical study but rather the procedures that have seemed most efficacious in my hands. In addition, certain types of congenital lens anomalies that warrant special consideration will be discussed.

Introduction

A thorough knowledge of congenital cataracts is essential for the intelligent discussion of the case with the patient or the patient's parents. It is also of importance in reaching a decision as to whether or not the cataract should be operated upon and the type of surgical procedure that may be indicated.

As Duane[2] points out, it is only in exceptional cases that the congenital cataracts are discovered immediately after birth, the rule being that they are not noticed until the child is some weeks or months old. This is due to the fact that newborn children have very narrow pupils, that they sleep so much, and keep their eyes closed most of the time. Moreover, they do not fix objects, an additional factor in not making it apparent that they do not see. Partial congenital cataracts may be overlooked for a longer time as the child will have sufficient vision to carry on.

Certain forms of congenital cataract show an hereditary tendency, primarily of a dominant type, although the type may not always be consistent. Therefore, lamellar, polar, spindle, and central cataracts all may occur in the same family. In some instances, however, the type may be the same in all members of the family involved. Haro,[3] for example, observed a family of 59 persons, covering four generations, in which 16 members had ring or disk-shaped cataracts. Nettleship[4] found coralliform cataract in 35 members of one family over five generations. Franceschetti[5] has listed as types with an hereditary tendency: Congenital total cataract, lamellar cataract, embryonal nuclear cataract, coralliform cataract, spear cataract, floriform cataract, axial fusiform cataract, stellate cataract and disk-shaped cataract.

Production of congenital cataracts

As Falls[1] pointed out, ophthalmologists, to a large extent, are ignorant of the primal causes of congenital cataracts, especially in regard to the mechanism of their production

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* Presented at the III Pan-American Congress of Ophthalmology, Havana, Cuba, January, 1948. From the Division of Ophthalmology, University of California Medical School, San Francisco.

and the rationale of their transmission from one person to another. Mann[6] states that a certain number of congenital abnormalities can be explained as abnormal persistence of a normally transient condition.

It must also be borne in mind, however, that a greater part of so-called developmental anomalies do not represent any normal stage, but are pure aberrations of growth. The most that can be said in regard to the production of these developmental anomalies is that it is possible to deduce from their location the time at which the fibers involved were formed. Whether the opacities occurred at this time or whether they occurred as the result of a subsequent degeneration, it is impossible to say.

Intra-uterine trauma may at times produce lens changes as seen in the reduplication cataract. In addition, as Mann[7] has stated, we know experimentally that toxic substances administered to the mother during pregnancy may cause lens changes. It is known that interference with calcium metabolism and parathyroid secretion will produce the same opacities produced by avitaminosis.

The recent work of Gregg,[8] Swan and his co-workers,[9] Reese,[10] and others on rubella cataract would tend to show that infection in the mother, particularly during the early part of pregnancy, is capable of producing lens changes.

In addition, lens changes may be secondary to abnormalities of the vascular capsule which produce such changes as retrolental fibroplasia (persistence and hyperplasia of the primary vitreous).

From the above it seems apparent that some knowledge of the embryology of the lens, together with a thorough knowledge of its structure, particularly the zones of discontinuity, is essential for the proper diagnosis of the type of congenital cataract. As already stated, the correct diagnosis is essential in helping to make a decision in regard to the surgery of the case at hand.

Other anomalies are present

It is noteworthy that in approximately 50 percent of the cases of congenital cataract other ocular anomalies are present. These include strabismus, nystagmus, aniridia, choroidal and retinal changes, microphthalmus, subluxation of the lens, dissociated ocular movements, eccentric fixation, colobomas of the lens and optic nerve, muscle paresis, anomalies of the hyaloid artery system, and other changes. Falls[1] in his series of 313 cases found that 52.6 percent of the patients had other ocular defects in addition to congenital cataract.

In addition to ocular defects, a fairly high percentage of these cases have associated disturbances of a constitutional variety, either congenital or metabolic in nature. There is also a fairly high incidence of mental retardation. According to Krause[11] the mental deficiences may not make their appearance until the child is four or more years of age. The opinion has been expressed that the mental retardation is due to failure to perceive visual sensations. On the other hand all of us have seen instances of bilateral congenital blindness, uncorrectable by surgery, that have progressed very well mentally. While it is true that in many instances some of these cases do apparently show a marked improvement in their mental condition after restoration of vision, it must be kept in mind that frequently this does not occur. From what has been said it is obvious that the prognosis in the care of congenital cataracts must be guarded. As early as 1805, Ware[12] stated: "Operation is not so certain a cure for cataract in children, as in persons of a more advanced age."

General indication for surgery

The general indication for surgery in these cases is a visual acuity that has been reduced to the point of interfering with the normal use and development of the eyes. Inasmuch as most congenital cataracts are stationary it may at times be advisable to

leave the patient alone if his vision is 20/40 or better. It would seem advisable to make a general rule that patients with 20/40 vision should not be operated upon unless the visual acuity is further reduced. Consequently, it is never wise to tell a patient that he should never be operated upon, as the visual acuity may change in some of these cases. The zonular (lamellar) cataract, for example, has a tendency to increase in density so that, while the visual acuity may be adequate in youth, the vision in middle life may be sufficiently reduced to warrant surgery.

Age at which to operate

There has been a good deal of variance regarding the age at which congenital cataracts should be operated upon. In 1805 Ware[12] said, "Children, again, some of whom are born with cataracts, are in general so unmanageable that the operation becomes almost impracticable. For these reasons it is advisable to postpone it until they arrive at the age of reason and reflection, and feel by experience the necessity of submitting to it." It must be remembered this was before the days of anesthesia.

By 1830 we find that the opinion has changed. At that time Mackenzie[13] says, "In cases of congenital cataract, ought the operation to be delayed till the patient has attained an age sufficient to enable him to give his assent, or ought it to be practiced during infancy? The answer decidedly is to operate in infancy. About the age of from 18 months to 2 years, the tissues of the eye have attained a degree of resistance which enables the surgeon to operate with greater precision than at an earlier period, yet the capsule is not so tough and coriaceous as at a later period, and especially after the lens (as often happens in congenital cases) is completely absorbed."

Guthrie[14] in his lectures on the operative surgery of the eye, in 1830 states: "The proper period for operating has been and ever remains a disputed point, not as to the propriety of doing it at an early age, or within the first three years, that being fully established; but as to the best part of that time, some surgeons recommending 6 months, others 20 months to 3 years. If the child be healthy at 3, 4, and certainly 6 months, the operation ought to be performed with every hope of success. If the operation is delayed until the end of the third year, the child acquires . . . an irregular rolling motion of the eye, which it does not readily lose."

E. Fuchs,[15] in 1928, in discussing congenital cataracts said: "When I was still an assistant in the clinic, it was the rule that children with congenital cataracts were not to be operated upon until they were 10 years old. It was only later that it was discovered that the earlier the operation the better the visual result. One should not expect to obtain normal vision in such eyes as they also show other evidences of abnormal development. These eyes are usually smaller than normal and, if one waits until later to operate, the eye will have increased some in size, but the visual acuity will be much worse because the retina, due to lack of use, fails to develop normally."

Kirby[16] states that if operation is decided upon, it may well be performed early, as good results are obtained with careful needling at the age of a few months. If the lens opacities are allowed to obstruct the rays of light until after the fourth year of life, the best function obtainable after their removal will not be within the range of useful vision. He also states that in progressive congenital cataracts the visual function of the macula may be well developed in the early period of life and therefore the function will be good after the lens has been removed at a later date. Such patients obtain good vision after operation as late as 9 to 10 years of age.

Falls[1] suggests that in cases of complete bilateral cataracts, operation be performed on one eye after the patient reaches the age

of six months and on the other after the age of two years. If the cataracts are incomplete and the child has sufficient vision to get around, he feels surgery should be postponed until the 3rd or 4th year. Treacher Collins[17] prefers to wait until the child is at least 10 months old, as at that age the eye is larger and the anterior chamber is deeper.

From what has been said it is apparent that there can be no hard and fast rule as to the time of surgery. It has been my practice to operate the first eye at six months in those cases where the cataract is sufficiently dense to interfere with fixation, in the hope that the resultant fixation will prevent the establishment of nystagmus. The second eye is operated upon between the 2nd and 3rd years. In the interim, the eye has had the opportunity to develop and increase in size so that surgery is simpler. When the lens opacity is sufficient to interfere with the possibility of the child learning to read, the cataract should be removed before the school years.

Resultant postoperative vision

The prognosis as to the amount of resultant postoperative vision must be guarded. Fuchs[15] states that, "A visual acuity of one-fourth of normal certainly seems rather poor but must be considered a good result in these cases, especially where there is nystagmus, a smaller eye, and an abnormal development of the retina. One learns to appreciate such a result when one is asked to operate a case of bilateral congenital cataract after puberty. Immediately following operation the patient appears as blind as he did before surgery for he does not recognize objects, due to the fact that he lacks visual memories. This must be developed through experience but even then he will not obtain more than defective vision because of the fact that after a certain age the retina cannot entirely overcome the neglected development. One reads in romances how the congenitally blind person is operated upon and how at the first removal of the bandages he is surprised at the beauty or ugliness of his adored-ones. This must be regarded as purely poetic fantasy."

In Falls's[1] series 53.5 percent had a visual acuity of 20/100 or better; 9 percent were capable of 20/200 vision or possessed the ability to place accurately the Worth ball. For all practical purposes 17 percent were blind; 18.4 percent were capable of reading J4 to J7 print or better. It is interesting that in his series Falls found that no patient with pendular (ocular) nystagmus achieved better than 20/50 vision.

From the above it is apparent that in discussing the surgery of congenital cataracts with the parents it is well to explain in detail the visual prognosis and thus avoid a great disappointment on the part of the parents.

Surgical procedures reported in literature

The surgery of congenital cataracts has been limited to a relatively few procedures, all of which have their advocates. The ideal surgical procedure for the removal of congenital cataracts would seem to be one in which a single operative procedure would provide for the removal of at least the major portion of the lens so as to permit the remainder to absorb without further surgical interference. In addition, there should be no interference with the pupil and a minimum danger of iris incarceration or adherence to the operative wound. It is also highly desirable that, if possible, there be no interference with the posterior capsule and the vitreous. The danger of glaucoma should be minimized.

The use of visual iridectomy as advocated by Fox[18] and others in cases of incomplete cataract has been discarded. Bell[19] advocated an iridectomy as a preliminary procedure to either a later Ziegler discission or a linear extraction.

Discission

Discission in one form or another is the

most universally used operation for congenital cataract. The simple discission, repeated a varying number of times, has many advocates because of its simplicity and the fairly good results obtained. One of the disadvantages, a very real one from the parents' viewpoint, is that the procedure must be repeated a number of times. While complications are not common, as Spaeth[20] points out, they do occur and include cessation of the absorption as a result of postoperative irritation; postoperative low grade and prolonged iridocyclitis; organization of capsular tags and remnants into a dense membrane. Secondary glaucoma also occurs. As Wiener and Alvis[21] stated, discission or needling as many as 17 or 18 times has been necessary before a good opening or good visual results were secured.

In 1921 Ziegler[22] published his V-shaped through-and-through discission of the lens, which has many advocates. Wiener and Alvis[21] say that it is rarely necessary to operate these patients more than once and practically never more than twice. They feel that there is absorption of the softened lens in the anterior chamber and in the vitreous, "thus equalizing the pressure within the eye." The chief objections to the operation are delayed absorption and the presence of vitreous in the anterior chamber. Spaeth[20] says that a through-and-through incision of the lens into the vitreous is not a guarantee against the development of secondary glaucoma; instead, damage may occur to the vitreous with later development of a chronic iridocyclitis and even a retinal detachment.

Modification of discission

Recently, Moncreiff[23] has reported his modification of the discission. He makes an incision through the anterior capsule and into the cortex, and then makes multiple incisions in all directions down to the nucleus until the anterior cortex and capsule are reduced to shreds. The nucleus is then cut into several fragments. With an anterior-chamber irrigator he attempts to wash all of the cortical material from the remnants of the capsular sac into the anterior chamber. He feels this is the nearest approach to the ideal operation in these cases. The operation has only been done in both eyes of three patients and, as Moncreiff states, a large number of cases will have to be observed before the operation can be evaluated. It would seem that the possibility of glaucoma, due to the rapid swelling of all of the lens material in the anterior chamber, would be a definite risk.

Another rather frequently used procedure is the combination of discission and linear extraction. A fairly extensive discission of the anterior capsule of the lens is done. The eye is observed for several days postoperatively until the lens material is sufficiently disintegrated to permit easy removal through corneal or limbal keratome incision. Falls[1] found that this combined technique produced the best visual results. One does, however, have to keep in mind that in the older age group of children this may produce an acute glaucoma following the discission.

Modification of linear extraction

In 1932, Otto Barkan[24] reported his modification of a simple linear extraction in which the cataract extraction was done in one procedure. The chief modification consisted in several very important details, namely, the injection of adrenalin at the limbus and the making of the keratome incision inside the limbus. The absorption is rapid, and useful vision is obtained in a very short time, much sooner than with the discission. The difference in time between this and the combined method of discission and linear extraction is not very great, however, but it does not necessitate two operations. The chief objections that have been made to the procedure have been the tendency to anterior-synechia formation and the possibility of vitreous loss. The avoidance of these rather infrequent complications will be discussed later.

The intracapsular extraction also has a

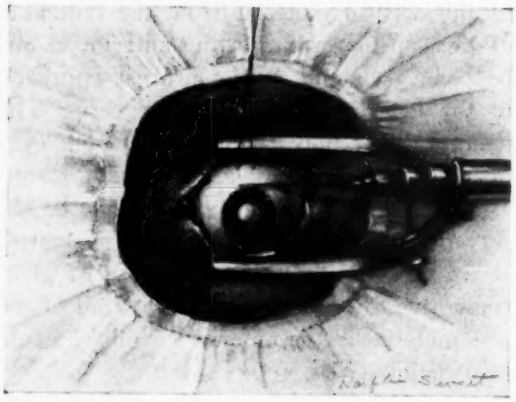

Fig. 1 (Cordes). Two drops of adrenalin (1:1,000) are injected subconjunctivally at the limbus above. When introducing the needle, the open part should be directed toward the limbus.

very limited use in congenital cataract surgery. Falls[1] in his series limited this operation to the treatment of shrunken, dense, membranous, or hard cataracts in the very young.

Results in various operations

Horay's[25] observation on the results in the various types of operation for congenital cataract is of interest. He reported the results in 282 operations on congenital cataract. The cases were divided equally between discission, linear extraction (simple), and combined linear extraction. The age of the patient played no part in the decision as to the type of operation to be used.

From the survey it was concluded that the best results were obtained with a combined extraction, while the poorest results were obtained with the discission. The opinion that has been expressed that the operation should not be done until after the first year he felt was unfounded as the results were just as satisfactory during the first year as later in life. The failures in the patients operated upon during the first year of life were often the result of other congenital anomalies present. He concluded that an extraction, done in one sitting, was the operation of choice, irrespective of the age of the child.

Surgical procedures in congenital cataract

Except for certain types of congenital cataracts to be discussed later, I have employed a slightly modified Otto Barkan operation on all cases.

After the routine preoperative preparation and anesthesia, two drops of adrenalin (1:1,000) are injected subconjunctivally at the limbus above (fig. 1). When introducing the needle, the open part should be directed toward the limbus. After 5 to 10 minutes the pupil is maximally dilated so as to almost disappear behind the limbus, especially above at the site of the incision. This dilation is due to a contraction of the sympathetic dilator fibers and thus is tonic, and results in maintaining the exaggerated dilation even after the anterior chamber is open, thereby preventing the iris from coming into contact with the incision before the anterior chamber is reformed. In addition, the hypotony is such that the tendency of the ocular

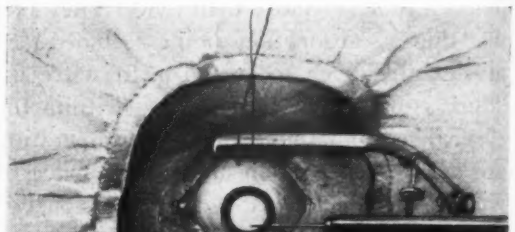

Fig. 2 (Cordes). Before making the incision, a puncture is made at the lower temporal limbus with a straight knife-needle to facilitate the injection of air into the anterior chamber after completion of the operation.

contents to prolapse is reduced to a minimum.

A suture is now placed through the superior rectus tendon. This is very helpful in fixing the eye downwards during the extraction, especially in infants where the small

area of the operative field makes it difficult to use fixation forceps.

Before making the incision, a puncture wound is made at the lower temporal limbus with a straight capsulotomy knife-needle to facilitate the injection of air into the anterior chamber after completion of the operation (fig. 2). The site of the puncture should be marked with gentian violet as an aid in finding it later. If the knife-needle is introduced into the chamber parallel to the iris surface, there will be no loss of aqueous

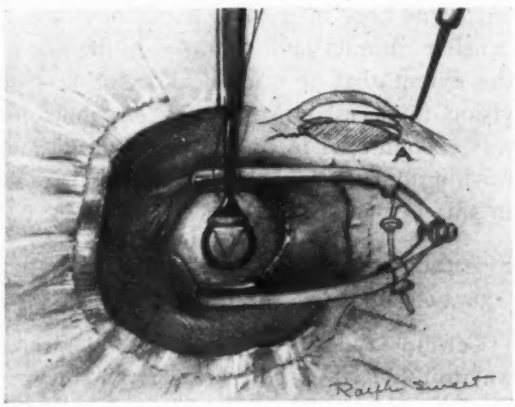

Fig. 3 (Cordes). A keratome incision is made inside the limbus and parallel to the iris plane.

and no collapse of the anterior chamber.

The incision is made 10 minutes after the injection of the adrenalin so as to allow ample time for the maximum effect. A keratome incision is made 1.5 to 2 mm. inside the limbus in the cornea. The incision is made oblique (parallel to the iris surface), thus forming a valvelike incision which tends to close more the greater the pressure within the anterior chamber (fig. 3).

The incision should be no larger than necessary for the extraction of the lens substance.

A capsulotomy is then performed with capsule forceps. As in the senile extracapsular extraction, it is desirable to obtain as large a capsulotomy as possible. In very young infants this must necessarily be small (fig. 4).

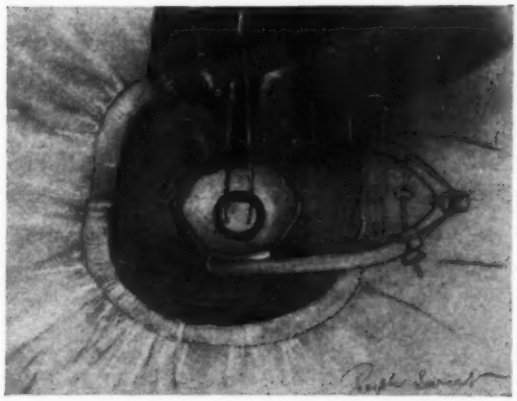

Fig. 4 (Cordes). A capsulotomy is done with Fuchs's forceps.

The lens substance is now expressed with spoons and the anterior chamber is washed out with normal saline solution (fig. 5). The wound is carefully inspected to be certain that there is no lens substance or capsule remnant in the wound.

With a small Luer syringe and a very fine needle (28 to 30 gauge), air is injected into the anterior chamber. This is done without difficulty through the small, previously made, stab puncture at the limbus below. The tip of the needle should be dulled a bit so that the point will not catch in the walls of the puncture wound. The injected air remains in the upper part of the anterior chamber for several days and prevents the iris from

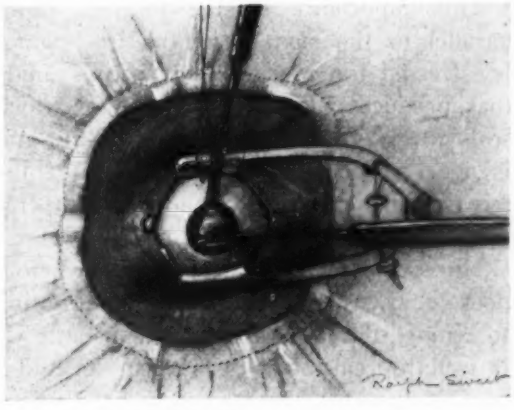

Fig. 5 (Cordes). The lens substance is expressed and the anterior chamber is washed out with normal saline solution.

coming into contact with the wound. Otto Barkan emphasizes the importance of a deep chamber before the patient leaves the operating table (fig. 6).

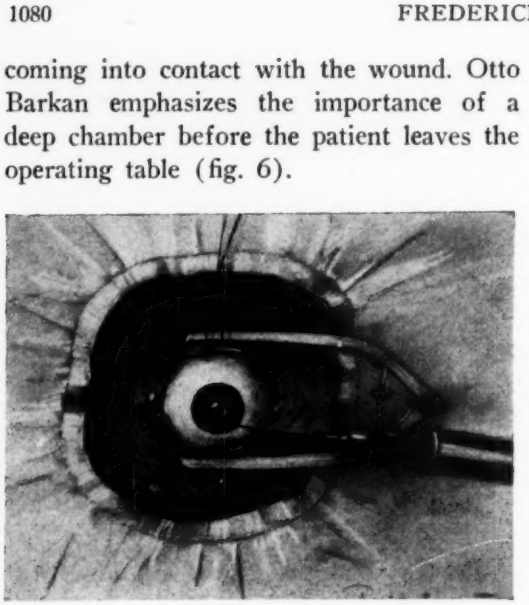

Fig. 6 (Cordes). With a small Luer's syringe and a very fine needle (28 to 30 gauge), air is injected into the anterior chamber. The tip of the needle should be dulled to facilitate the introduction of the needle into the previously made canal.

The usual postoperative atropinization is carried out.

Complications

The complications encountered are not many and can, to a great extent, be prevented. Of importance is the injection of adrenalin and the necessary wait of 10 minutes until the pupil is widely dilated so that the iris is not in the way.

The importance of the incision being parallel to the surface of the iris cannot be stressed too much, for it is this angle that produces the valvelike opening which seals firmly from the pressure within, and is a factor in preventing prolapse of the iris or opening of the wound by postoperative squeezing.

Vitreous loss is infrequent and, when it does occur, usually results from attempting to remove the last bit of lens substance. It is well to remember that when the major part of the lens has been removed there is little to absorb and this absorption occurs in a short time, even in those cases where the lens is relatively immature. It is important not to "do too much."

The most serious objection that has been made to the operation is the development of anterior synechias, or iris incarceration in the wound, with the resultant drawing up of the pupil which may at times be accompanied by glaucoma. The injection of air immediately following the operation seems to prevent this, as in no instance where this has been done has there been any iris incarceration.

Secondary glaucoma has occurred only once, and this in a 12-year-old boy whose lamellar cataract had increased in density to the extent that it caused sufficient loss of vision to necessitate surgery. No difficulty was encountered in opening the wound and washing out the lens debris. The final result in this eye was 20/30 vision.

Special consideration of certain types of congenital cataracts

Certain types of congenital cataracts warrant special consideration from the surgical viewpoint.

Posterior polar cataract. In this cataract, in addition to the posterior cortical opacity, the major opacity is confined to the posterior capsule and therefore, if a linear extraction is done, a later discission through this posterior capsule is necessary. Due to the fact that other anomalies are often present the prognosis must be guarded when surgery is considered.

Embryonal nuclear cataract. It must be remembered that in these cases the nucleus is dense and hard so that discission is very unsatisfactory. The cataract, however, causes very little interference with vision and Gifford[26] goes so far as to say that operation is never required for opacities limited to the embryonal nucleus.

Zonular (lamellar) cataract. In some of these cases, the disturbance continues until birth, or, is so unusually severe that the entire lens is opaque. Under these conditions

further growth of the lens fibers does not take place. This type of zonular cataract is usually associated with other serious congenital defects of the eye so that operative prognosis must be guarded.

Membranous cataract. In the membranous cataract, with the absorption of the lens parenchyma during intra-uterine life, the anterior and posterior capsules collapse to form a thin gray-white or chalky-white membrane. The condition is frequently bilateral and often associated with other anomalies including nystagmus and strabismus. In these cases the use of the discission advocated by Wheeler[27] has been found simple and effective.

Axial fusiform cataract. It is well to remember that in the axial fusiform cataract actual adhesion of the nucleus to the anterior and posterior capsules has been demonstrated.

Disk-shaped cataract. The surgical treatment of this cataract presents an interesting problem, due to the fact that for all practical purposes there are two types of cataract in one individual, namely a membranous (central area) and a soft cataract (the periphery of the lens). The literature contains relatively little on the subject.

Collins[28] was able to separate the central plaque in one of his patients. In both eyes the plaque fell into the lower part of the anterior chamber, where it remained without causing any reaction. Haro[3] reported his surgical experience in two cases. In one patient he attempted two discissions with rather poor results. After absorption of the lens substance, the central membrane was cut by means of de Wecker's scissors through a keratome incision. In the second case three discissions were performed but they were only partially successful. In the fourth operation, de Wecker's scissors were used to cut through the central membrane.

The first operation in these cases should be a discission through the existing lens substance and through the membrane, thus allowing the lens fibers to absorb. As Haro[3] has pointed out, in most cases the membrane is firmly adherent to the rest of the lens and is too tough to be cut with an ordinary discission needle. For this reason it would seem advisable to use the Wheeler knife-needle and to do the procedure in the manner described by Wheeler.[27] If this does not succeed it would seem that the use of the de Wecker or Berens scissors would be the next step.

Total extraction must be considered with caution as in some of these cases the central portion of the membrane is adherent to the hyaloid and extensive vitreous loss may follow this procedure as described by Malbran and Tosi.[29]

Rubella cataract. This type, which has been recognized as an entity in the last few years, deserves special consideration. It must be remembered that these cases often have other congenital anomalies and that microphthalmus is a common finding and therefore the prognosis must be guarded.

As Reese[10] has stated the anterior chamber is particularly shallow and in many cases the very dense central portion of the lens has proved resistant to the needle, sometimes separating off as a firm disk. If needling is employed this disklike opacity should be removed from within the confines of the capsule and, if possible, broken up to rest in the anterior chamber where it will absorb.

This characteristic of the embryonal nucleus in some of these cases has been observed by Goar and Potts[30] and others. In some of the cases the entire lens is opaque and here the usual operative procedure is employed. We have employed linear extraction as described above in these cases and have encountered only the complications of other anomalies seen in these cases.

Absorption of the lens substance has been slower than that of the usual congenital cataract. In most of these cases it is difficult to dilate the pupils with any mydriatic and this is especially true after operation, so

that in a certain percentage, there is an occlusion of the pupil in spite of all attempts at mydriasis. Glaucoma is one of the complications encountered.

Ether anesthesia has been used in these cases in spite of the presence of heart lesions. Goar and Potts,[30] Reese,[31] and Ellett[32] all agree that these patients stand general anesthesia well in spite of the presence of the heart lesion. Post[33] in one case used local anesthesia without difficulty. Cocaine was used as instillations as for a needling operation. The baby was firmly held and the operation performed on each eye without apparent pain to the infant and no operative difficulties were encountered.

The prognosis must always be guarded in these cases, as it is impossible to state whether or not other ocular defects are present or will develop.

Persistence and hyperplasia of the primary vitreous (retrolental fibroplasia). While this condition is, strictly speaking, not a cataract, it is associated with the lens and often is accompanied or followed by lens opacities and therefore warrants discussion here. On the theory that the condition resulted from failure of production of the aqueous, Terry[34] attempted to establish new vascular connections between the ciliary body and the episclera by scleral trephination over the ciliary body and reported some improvement in this condition.

The most important work on the subject is that done by Reese and Payne.[35] On a premise that some of these eyes might see, they made an effort to salvage 6 eyes in 6 cases. Their procedure was to remove the lens substance by repeated discissions and finally to make a vertical cut in the retrolental fibrous tissue with de Wecker's scissors.

Very little seems to happen when the lens capsule is opened, so that the capsule opening can be made extensive without fear of undue or too rapid swelling. After the lens substance had been dissipated for the most part, a central vertical incision was made through the remainder of the tissue with de Wecker's scissors. This was followed by hemorrhage in the anterior chamber with absorption in the due course of time.

In one instance they used three discissions and two incisions with de Wecker's scissors. In 2 of the 6 cases there was some vision, which it was believed came from the operated eye. While the results, according to the authors, leave a lot to be desired, the procedure may develop into one that may be helpful in these otherwise desperate cases.

Cataracts following intra-uterine inflammation. The prognosis is very bad in those cataracts with firm posterior synechias resulting from intra-uterine inflammation. Experience has shown that any operative procedure in these cases is "invariably followed by inflammation and fibrosis."[30]

Summary

In the surgical treatment of congenital cataract, it is important to remember that 50 percent of the patients have other ocular congenital anomalies and that therefore the prognosis must be guarded. In addition other general congenital anomalies are often present.

As a rule a visual acuity of 20/40 or better would seem to be a sufficient contraindication for surgery inasmuch as the wearing of the heavy disfiguring lenses is in itself a definite handicap to the growing child. In some instances, as in the lamellar cataract, the opacity may increase in density so that later surgery is necessary.

From personal experience, and from the literature, it would seem that in those cases of bilateral congenital cataract where the opacities are sufficiently dense to interfere with the development of fixation, the first eye should be operated upon at six months so that the child may develop this faculty and thus avoid nystagmus. The second eye should be operated upon between the 2nd and 4th year. When the cataracts are of

sufficient density to interfere with the child learning to read, they should be operated upon before the school age.

In discussing surgery of congenital cataracts with the parents, it is well to remember that only approximately 50 percent of the cases develop a postoperative vision of 20/100 or better.

The personal choice of operation in soft congenital cataracts is the linear extraction as modified by Otto Barkan. The important modifications are the injection of adrenalin (1:1,000) at the limbus at the site of the incision, to prevent the iris from becoming involved; and the making of the incision parallel to the iris surface and 1 to 2 mm. inside the cornea. In addition it has been found that the injection of air into the anterior chamber at the completion of the operation is an important factor in the prevention of the formation of anterior synechias or incarceration of the iris. The importance of a deep anterior chamber when the patient leaves the operating room has been stressed by Barkan and cannot be emphasized too much.

In certain types of congenital cataract, the discission as advocated by Wheeler is indicated, particularly in the congenital membranous cataract.

In the disk-shaped cataract where there are two types present (membranous central portion and peripheral soft cataract), a discission followed later by an incision through the membrane with Berens or de Wecker scissors is usually necessary.

In rubella cataract, in addition to the possibilities of other anomalies being present, it is well to remember that the absorption of the lens substance is slower than that of the usual congenital cataract and that in most instances it is difficult to dilate the pupil with mydriatics.

In persistence and hyperplasia of the primary vitreous it is possible that repeated discission of the lens substance, finally followed by making a vertical cut in the retrolental fibrous tissue may offer some hope in these otherwise hopeless cases.

The surgical treatment of congenital cataracts is a grave responsibility as the outcome of the operation is instrumental in determining the individual's future position in society. A thorough knowledge of the types of congenital cataracts, combined with a detailed study of the case in hand, is important in making the decision as to whether or not surgery is indicated. If surgery is indicated, this knowledge is useful in determining the age at which the operation is to be performed and the type of surgical procedure to be employed.

384 Post Street (8).

References

1. Falls, H. F.: Developmental cataracts. Arch. Ophth., **29**:210-223 (Feb.) 1943.
2. Duane, A. in Fuchs, E.: Textbook of Ophthalmology. Philadelphia, J. B. Lippincott Co., Ed. 7, 1923, p. 638.
3. Haro, E.: Hereditary disk-shaped cataract: Report of a family with microscopic examination of an eye. Arch. Ophth., **36**:82-100 (July) 1946.
4. Nettleship, E.: On some hereditary diseases of the eye. Tr. Ophth. Soc. U. Kingdom, **29**:57, 1909.
5. Franceschetti, A.: Cataracta congenita. Kurzes Handb. d. Ophth., Berlin, Julius Springer, vol. 1, 1930, p. 743.
6. Mann, Ida C.: The correlation of the embryology of the lens with slitlamp appearances. Tr. Ophth. Soc. U. Kingdom, **45**:696 (Part II) 1925.
7. ———: Developmental Abnormalities of the Eye. Cambridge: The University Press, 1937, p. 335.
8. Gregg, N. M.: Congenital cataract following German measles in mother. Tr. Ophth. Soc. Australia (1941), **3**:35-46, 1942.
9. Swan, C., Tostevin, A. L., Moore, B., Mayo, H., and Black, G. H. B.: Congenital defects in infants following infectious diseases during pregnancy. M. J. Australia, **2**:201-210 (Sept.) 1943.
10. Reese, A. B.: Congenital cataract and other anomalies following German measles in mother. Am. J. Ophth., **27**:483-487 (May) 1944.

11. Krause, A. C.: Congenital encephalo-ophthalmic dysplasia. Arch. Ophth., **36**:387-444 (Oct.) 1946.
12. Ware, James: Chirurgical observations relative to the eye: With an appendix on the introduction of the male catheter and the treatment of hemorrhoids. Printed for J. Mawman, in the Poultry, London, 1805, vol. 2, p. 2.
13. Mackenzie, William A.: A Practical Treatise on the Diseases of the Eye. London, Longman, Rees, Orme, Brown & Green, 1830, p. 596.
14. Guthrie, G. J.: Lectures on the Operative Surgery of the Eye. London, Burgess and Hill, 1830, p. 389.
15. Fuchs, E.: Festschrift der Konigl. Universitata-Augenklinik in Zagrez. Jugoslavia, Zagreb, 1928, p. 80.
16. Kirby, D., in Berens, C.: The Eye and Its Diseases. Philadelphia, W. B. Saunders Co., 1936, p. 565.
17. Treacher Collins, E.: Developmental deformities of the crystalline lens. Ophthalmoscope, **6**: 577-663, 1908.
18. Fox, L. W.: Congenital cataract: Plea for variety in its surgical treatment. J.A.M.A., **89**:2249-2252 (Dec.) 1927.
19. Bell, G. H.: Operation for juvenile cataract in two stages. Am. J. Ophth., **9**:433-435 (June) 1926.
20. Spaeth, E. B.: The Principles and Practice of Ophthalmic Surgery. Philadelphia, Lea & Febiger, Ed. 3, 1944, p. 612.
21. Wiener, M., and Alvis, B. Y.: Surgery of the Eye, Philadelphia, W. B. Saunders Co., 1939, p. 54.
22. Ziegler, S. L.: Complete discission of lens by V-shaped method. J.A.M.A., **77**:1100 (Oct.) 1921.
23. Moncreiff, W. F.: Surgery of congenital cataracts. Am. J. Ophth., **29**:1513 (Dec.) 1946.
24. Barkan, O.: Procedure for extraction of congenital, soft and membranous cataracts. Am. J. Ophth., **15**:117-124 (Feb.) 1932.
25. Horay, G.: Ueber Staroperationen im Kindesalter. Klin. Monatsbl. f. Augenh., **74**:178-184 (Jan.-Feb.) 1925.
26. Gifford, Sanford: Textbook of Ophthalmology, Philadelphia, W. B. Saunders, Ed. 3, 1945, p. 211.
27. Wheeler, J. M.: Secondary cataract opening by single straight incision: Iridotomy by same method. Am. J. Ophth., **8**:179 (Mar.) 1925.
28. Treacher Collins, E.: On the pathogenesis of anterior polar cataract. Tr. Ophth. Soc. U. Kingdom, **18**:124, 1898.
29. Malbran, J., and Tosi, B.: Cataracta anular congenita. Arch. de Oftal. de Buenos Aires, **15**:543-558 (Nov.) 1940.
30. Goar, E. L., and Potts, C. R.: The relationship of rubella in the mother to congenital cataracts in the child. Tr. Am. Ophth. Soc., **43**:135, 1945.
31. Reese, A. B.: Discussion of Goar and Potts' paper. Tr. Am. Ophth. Soc., **43**:141-142, 1945.
32. Ellett, E. C.: Discussion of Goar and Potts' paper. Tr. Am. Ophth. Soc., **43**:144-145, 1945.
33. Post, L. T.: Discussion of Goar and Potts' paper. Tr. Am. Ophth. Soc., **43**:143-144, 1945.
34. Terry, T. L.: Retrolental fibroplasia in premature infants; Further studies. Arch. Ophth., **33**: 203-210 (Mar.) 1945.
35. Reese, A. B., and Payne, Frank: Persistence and hyperplasia of the primary vitreous. Tr. Am. Ophth. Soc., **43**:163-192, 1945.

PREVENTION AND MANAGEMENT OF COMPLICATIONS OF CATARACT EXTRACTION*

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A comprehensive review of complications of cataract extractions was published by Berens and Bogart,[1] in 1938, and this study was supplemented by Kirby[2] in his 1941 paper on the practical aspects of both operative and postoperative complications.

Statistical analyses of the results and of the complications from senile cataract extractions performed in the Wilmer Institute between 1925 and 1943 have been recorded in careful detail by Hughes and Owens.[3, 4] They were able to divide the extractions performed during that period into five main types, with each subsequent type giving better results than the preceding one: (1) Combined extracapsular extractions with corneal sections and no sutures, (2) combined extracapsular extractions with conjunctival flaps and sutures, (3) combined intracapsular extractions with conjunctival flaps and sutures, (4) combined intracapsular extractions with one (rarely two) corneoscleral sutures, and (5) intracapsular extractions with peripheral iridotomy and two (rarely one) corneoscleral sutures. The last type of extraction gave significantly better final results than any of the first four types.

The statistical comparison of these groups is subject to one criticism; namely, each of the five types of extraction was in successive general preference during five successive eras of the period covered by the study and, therefore, minor refinements in operative technique, preoperative care, and postoperative care may have accounted for some of the improvements in results. Nevertheless, the conclusions of Hughes and Owens are certainly valid in general and the procedure of choice for extracting senile cataracts is intracapsular with peripheral iridotomy and two corneoscleral sutures.

This present report purposes to supplement the studies of Hughes and Owens by elaborating upon details of cataract extraction which do not lend themselves to ready statistical analysis. In most instances, the procedures herein advocated represent current preferences of the Wilmer Institute staff as a whole, such procedures having come into general usage as a result of cumulative experience.

PREOPERATIVE MANAGEMENT

PREVENTION OF PSYCHOLOGIC COMPLICATIONS

Prior to operation the patient should be told exactly what he must go through, what are the chances of a successful extraction, and what he can expect in terms of vision provided the operation is anatomically successful.

He should understand that (1) aphakic vision is so different from phakic vision as to require an appreciable period of adjustment (longer in elderly patients than in younger ones), (2) an aphakic eye can rarely ever be used together with a phakic eye with a correcting lens for each eye, (3) an aphakic eye is devoid of accommodation, and (4) when the initial correction is obtained for his aphakic eye he will be bothered by magnification, causing objects to appear closer than they really are, by peripheral distortion, and by limitation of the effective visual field. Limitation of effective field necessitates turning the head rather than the eyes. It is a permanent handicap, probably

* From the Wilmer Ophthalmological Institute of The Johns Hopkins Hospital and University. Presented before the Section on Ophthalmology of the American College of Surgeons, New York, New York, September 9, 1947.

the most unpleasant feature of aphakic vision.

The patient should be told of any abnormality, such as senile macular degeneration, which would impair the visual result, or of the possibility of any suspected but non-visualized abnormality such as retinal detachment.

Choice of anesthetic

Local anesthesia is used except in special instances. Nembutal (0.1 gm.) is given by mouth two hours, and again one hour, before operation. Homatropine (2-percent solution) is instilled one hour before operation. Cocaine (4-percent solution) and neosynephrin (10-percent solution) are instilled alternately very two minutes preceding operation. About 5 minutes before operation, akinesia of the orbicularis is obtained with an O'Brien or van Lint block. My personal preference is for a slightly modified O'Brien block: This consists of injecting 4 cc. of 4-percent novocaine containing adrenalin (1:20,000) through a 2.0 cm., 25-gauge needle into both deep and superficial layers at the site preferred by Benedict, which is 1.5 cm. from the middle tragus on a line from the middle tragus to the upper lip. A retrobulbar injection of 1.5 cc. of 4-percent novocaine and 1:20,000 adrenalin is given deep within the muscle cone, using a 3.5 cm., 22-gauge needle.

A general anesthetic is indicated if the eye is unduly prominent or if there is increased intraorbital tension (resistance of the globe to being pressed back into the orbit). In these cases, a general anesthetic reduces the chance of loss of vitreous. General anesthesia is also indicated for uncooperative adults, and is of course necessary for children. Sodium pentothal is preferable in adults and ether in children.

Prevention of infection

Routine conjunctival cultures are of doubtful practical value, but care is taken that the lacrimal drainage apparatus is patent, that there is no clinically apparent conjunctivitis or blepharitis, and that there is no upper-respiratory infection.

Prevention of infection is dependent largely on the care with which the operative field is prepared, provided aseptic operative technique (including the use of drapes, gloves, gown, mask, and cap) is followed. Our present routine procedure is as follows: Two hours before operation, and again one hour before, argyrol (20-percent) is instilled (to coagulate mucous) and the conjunctival sac is irrigated with normal saline. The lashes are cut, the skin and brow are scrubbed with soap and water, the conjunctival sac is irrigated copiously with saline, and the lid margins, skin, and brows are painted with tincture of iodine followed by alcohol. Iodine is preferred because we found cultures of the skin of the lids at the close of operation rarely showed any growth when tincture of iodine was used, but frequently showed growth (usually staphylococci) when either tincture of merthiolate or tincture of metaphen was used.

Choice of operative techniques

The Parke lid speculum is most satisfactory, especially because a superior rectus bridle suture can be attached to the post on the upper arm of the speculum; more secure and dependable fixation is attained with a suture adjusted in this way than if it is clamped to the drapes or held by the assistant.

For uncomplicated cases a shallow incision, with a Graefe knife, of between two fifths and one half the circumference of the cornea is preferred. A mature or almost mature lens with a dense nucleus requires a slightly larger section than does an immature or hypermature lens. Also, in a very old patient the lens is generally larger and requires a larger incision than in a younger patient.

If there are many cornea guttata (endothelial dystrophy) there is an appreciable chance of persistent epithelial edema after operation. In these cases I believe the possibility of this postoperative complication is re-

duced by use of a small, deep section, and that such a section is indicated even though there is an increased risk of postoperative hemorrhage. Severe or prolonged diabetes, or purpura, indicates a section in clear cornea. A small incision with a knife or keratome, and enlarged with scissors, is preferable if the anterior chamber is very shallow.

If there is an elevated intraocular pressure which does not fall after the retrobulbar injection of novocaine and adrenalin, a posterior sclerotomy should be done before the incision is made. If there is an obliterated anterior chamber, either of two procedures may be used. The first, which I prefer, is the injection of air into the anterior chamber, followed by the usual Graefe section. The second, more generally used, is an incision ab externo with the Lundsgaard knife, enlarged to proper size with scissors.

If there has been an external filtering operation for glaucoma, with a filtering bleb at the 12-o'clock position, the incision may be made: (a) through the filtering bleb, (b) in clear cornea in front of the filtering bleb, or (c) below. I prefer a Graefe incision beneath a prepared flap below, with two corneoscleral sutures. In the presence of a luxated or dislocated lens, the incision is usually best made above and the method of delivering the lens varied according to the position of the luxation.

Two corneoscleral sutures appear necessary for optimal closure of the incision, and more than two are usually redundant. Sutures inserted before section (preplaced) are preferred. Corneoscleral sutures inserted immediately following section (postplaced) shortens the time required for operation, but coaptation of the incision does not appear to be as uniformly perfect as with preplaced sutures. The Lindner[5] or McLean[6] sutures give tighter closure than the Stallard[7] type of suture.

McLean sutures are preferred by most members of the Wilmer Institute staff. I personally like a slight modification of the Lindner or McLean suture: This modification consists in making a partially penetrating incision or slot in the limbus, passing the sutures through from the corneal surface, across the slot and out the scleral surface and overlying conjunctiva. The needle should be carried across the slot in a radial direction, with the point being first engaged in the cornea about 1.5 mm. in front of the slot, passing across the slot at a depth of 0.2 to 0.3 mm., and emerging from the sclera about 1.5 mm. behind the slot.

A round-pupil extraction with one peripheral iridotomy at the 12-o'clock position, is planned for all cases except those (1) with active anterior uveitis at the time of operation, (2) with an inflammatory membrane covering part of the pupillary space, (3) with marked fibrosis of the iris, (4) where a biopsy of the iris is desired, (5) where glaucoma is also present, or (6) where vitreous is already present in the anterior chamber (as with dislocated lenses). In these instances a complete iridectomy is performed. If congestive glaucoma is present, the iridectomy is made large and basal. Posterior synechias are broken loose with a spatula before the lens is delivered. If there is a postinflammatory pupillary membrane, it is separated from the lens with a spatula and excised with a blunt-pointed scissors; a radial iridotomy is performed at the 6-o'clock position before the lens is removed. Separation of inflammatory membrane from the lens is best accomplished, without rupture of lens capsule, if a smooth cyclodialysis spatula is used; this is true even if there has been a severe inflammation such as sympathetic ophthalmia.

An intracapsular extraction is attempted in every case except that of a traumatic cataract with the capsule already ruptured. Congenital cataracts in children are not generally supposed to be suitable for intracapsular extraction. However, while the zonule is quite strong, the capsule is also generally very strong, and contrary to the usual custom I prefer to attempt intracapsular extraction. The lens is grasped near the lower equator with forceps and pulled hard enough to stretch the ciliary processes and bring the

lower zonule into view; the zonule can then be stripped from the lens with an iris spatula as the lens is tumbled. No undue reaction appears to result from thus stretching the ciliary processes.

The incision

With preplaced corneoscleral sutures, it is important for the surgeon to have forceps in one hand for any necessary handling of the sutures as the section is completed. The assistant should therefore fix the globe. He should grasp the bulbar conjunctiva near the lower limbus with curved fixation forceps and pull the tips of the forceps gently downward, not pressing backward, and avoiding any pressure on the globe. A completely untrained assistant can perform this fixation without danger, if he is trustworthy. He should be directed simply to hold the fixation forceps in the one desirable position. The surgeon himself can then release the forceps after he completes the section.

Preplaced sutures predetermine the plane of the section and actually make a Graefe section easier than if no sutures are present. A sharp point on the knife is necessary for a proper Graefe incision. One with a double-edged point facilitates both engagement and counterpuncture. The knife is engaged at the limbus, in the same position as the slot for the preplaced sutures. The blade is pushed across the anterior chamber just in front of the iris until the tip begins to disappear behind the opposite limbus. The tip is then elevated anteriorly until it engages the cornea, the counterpuncture is made, and the section is completed. Care is taken to follow the slot and not to tilt the knife blade. The knife is carried past the preplaced sutures one at a time, the slot being spread at each of these points by lifting the suture close to the corneal lip as the knife goes by.

Intracapsular extraction of the lens

Extraction of the lens with capsule intact depends in large measure on the instrument with which the lens is grasped. Forceps are preferred in all cases except for dislocated lenses or where the capsule is so tense it cannot easily be grasped.

In my opinion the best forceps are Castroviejo's adaptation of Arruga tips to Sinclair's cross-section forceps. However, the presently available forceps of this type vary considerably in strength, curvature of shank, size and shape of blades ("tips"), and coaptation of blades. In the same hands, some forceps rupture a large percentage of capsules, while other supposedly identical forceps rarely break one.

If the cross-action spring closes the blades too tightly, the capsule is likely to be torn, while too little tension will often allow the capsule to slip out of the grasp of the blades. Forceps exerting a pressure of about 150 grams between the blades are most satisfactory.

The blades should have no superior lips, so the capsule can be grasped beneath the lower border of the pupil without the iris being caught. Blades about 3 mm. in length are optimal. Perfect apposition of the blades on closure, with heel and tip of each blade meeting in unison and maintaining an evenly distributed tension on complete closure, is essential.

The blades must have no sharp edges and no tiny nicks or spurs. When closed on a calling card they should slide smoothly both vertically and horizontally, with about the same resistance to sliding in either direction, and should not scratch either surface of the card. The blades should be inspected occasionally with a slitlamp for tiny defects. If such defects are found, these should be smoothed out with rouge paper or the forceps should be discarded.

In the delivery of a lens with forceps, the pupil should be moderately or widely dilated. With a moderately dilated pupil, the closed blades are inserted beneath the iris at the 6-o'clock position, opened, and closed on the capsule in a position two-thirds the distance from the anterior pole to the equator. During this maneuver the end of the forceps

handle is held with thumb and forefinger of the left hand, and the opening and closing of the cross-action spring (which requires appreciable pressure) is worked with the right hand. The right hand is then readjusted so the forceps fit in it much as does a pencil, and have no tendency to wobble even with the fingers almost completely relaxed. The left hand is then removed and used to manipulate the Arruga expressor.

The forceps blades are lifted anteriorly and upward to the center of the cornea and the strength of the lower zonule is actually felt. The actual grasping of the capsule and this movement of the forceps often initiates rupture of the zonule. If it does not, rupture of the zonule is started by point pressure on the tense zonule just below the lower limbus.

Once dislocation has begun, the curved portion of the expressor is used to press the lower portion of the cornea against the junction of the lower border of the lens and the vitreous. By a combination of traction on forceps and counterpressure directed more upward than posteriorly, the lower border of the lens is tumbled upward into the incision. As the lens is tumbled, the forceps are rotated backward in order to keep the blades parallel to the grasped surface of the lens. Often a gentle side to side motion of the forceps assists in breaking the zonule along the sides as the lens is tumbled. After the lens is tumbled it is lifted out of the incision with the forceps, while the expressor, which is now actually behind the lens, is used to press the cornea upward and forward against the lens to complete delivery.

Occasionally with a very soft eye and a very anterior incision, final delivery of the lens can be facilitated by having the assistant press gently backward on the scleral lip of the incision until the lens is halfway through the incision. Also, should the upper zonule rupture before the lens is tumbled (a rare occurrence), the lens can more easily be pulled out without tumbling if the assistant retracts the upper iris with a spatula or blunt hook. However, if the assistant is not thoroughly trustworthy, it is better to exert more traction with the forceps in lieu of having him execute these maneuvers.

Use of an erisophake is indicated when the capsule cannot easily be grasped with forceps, and for dislocated lenses. One bugbear about erisophakes should be dispelled: Some surgeons fear that if the erisophake should slip, vitreous may be sucked out. Actually, formed vitreous will not pass through the lumen of an erisophake even if tremendous negative pressure is applied.

Of the various suction devices, my personal preference is for the Dimitry erisophake because of its simple light construction, and the fact that the amount of negative pressure can be varied. The more fragile the capsule, the less suction should be used. The tip or suction cup of any erisophake should have no sharp edge and should be free of nicks or spurs, just as with forceps.

For removal of a cataract which is not luxated, the erisophake is manipulated in much the same way as forceps, grasping the lens in the same position and tumbling it with a combination of traction and counterpressure. In cases of partially dislocated lenses, the erisophake is applied adjacent to the dislocation (whether above, below, or on the side) and the lens delivered either by tumbling or by sliding, depending upon the site of the dislocation. This maneuver may be assisted by the insertion of a spoon behind part of the lens after it has been lifted anteriorly with the erisophake.

Operative complications

Probably the most frequent cause of disastrous complications in cataract extractions is nervous tension on the part of the surgeon. Some degree of nervousness is a perfectly natural reaction of any surgeon faced with the responsibility of a patient's vision, but it generally decreases as self-confidence is gained.

A number of authors have emphasized the importance of nervous tension to the point

even of laying down rigid rules of conduct for the operator. However, no set rules can apply to all individuals. Some surgeons are more at ease after much sleep, others after little sleep; much physical exercise (especially shortly before an operation) usually increases nervousness, but occasionally has the opposite effect; many surgeons are made tense by caffeine and therefore do not drink coffee on operating days, while others are calmed by caffeine. These and many other factors can only be evaluated by each man.

Observation by a colleague frequently causes some nervous tension, if the surgeon has great respect for the observer's opinion, and observation by a relative of the patient will often have the same effect. The surgeon is usually more at ease if he sits down and makes himself as comfortable as possible while operating, takes advantage of every available rest to steady his hands while undertaking the more delicate maneuvers, and uses those techniques for cataract extraction he believes will in his hands give the least chances of complications. Thus a surgeon who does not feel confident with a Graefe knife can make equally satisfactory incisions, at the expense of very little additional time, by the use of keratome and scissors; and one who is not ambidextrous can use the better hand for performing the section or grasping the lens in either eye.

The complications of cataract extraction which actually become apparent during operation are as follows, in order of their importance:

A. Vitreous loss

Loss of strictly fluid vitreous is of no consequence. Such loss can generally be inferred rather than seen. It is manifested by immediate reformation of the anterior chamber as the lens is delivered. In most eyes with "fluid" vitreous only the anterior vitreous is strictly fluid (by virtue of partial collapse posteriorly of the normal vitreous structure, or sheets), and if more than a certain amount of the truly fluid vitreous escapes from the eye, formed vitreous will then begin to emerge.

Loss of any formed vitreous causes some degree of vitreous opacities, renders impossible the preservation of a round pupil, and greatly enhances the probabilities of postoperative inflammation ("uveitis"), of postoperative glaucoma, or of late detachment of the retina. These undesirable sequelae follow in more or less direct ratio to the amount of formed vitreous which is lost. By "formed" vitreous is meant any vitreous which possesses recognizable structure, as contrasted with strictly "fluid" vitreous, which is indistinguishable from aqueous except for being slightly more viscous.

The unfortunate sequelae of vitreous loss are dependent not on the eye's actually being devoid of some vitreous, but on distortion of the remaining vitreous structure and especially on incarceration in the incision of the remaining vitreous sheets, which are still attached to the posterior segment of the eye. This incarceration results in delayed complete healing of the incision, with connective-tissue proliferation (and subsequent contracture) extending from the incision for some distance along the vitreous sheets. This fibrosis may block part of the angle, irritate the adjacent ciliary body, and exert traction on the retina via the vitreous sheets.

Vitreous loss is very seldom encountered if there is no pressure on the eye. Thus in a "soft" eye there is little or no danger of vitreous loss unless there is grossly improper counterpressure on the eye, or actual instrumental removal of vitreous. Although some surgeons have expressed a dislike for a "soft" eye because of difficulty in delivering the lens, this has not been our experience.

Avoidance of pressure depends on (1) akinesia of the orbicularis, (2) akinesia of the extraocular muscles, (3) an adequate palpebral fissure, and (4) lack of excessive retrobulbar bulk.

1. Complete akinesia of the orbicularis can be attained in a very high percentage of

cases by the slightly modified O'Brien block. Closure of the lids is tested just prior to beginning the operation; should akinesia be imperfect, a van Lint injection of novocaine should be given just outside the lateral orbital margin.

2. Complete akinesia of the extraocular muscles is only occasionally attained with a retrobulbar injection of novocaine, but partial akinesia is the rule if the injection is given deep in the muscle cone. In a series of cases where 1 cc. of 2-percent pontocaine solution was used for retrobulbar injection, better akinesia resulted than with 4-percent novocaine. However, such injections of 20 mg. of pontocaine are probably inadvisable as a routine procedure because of the very occasional idiosyncrasy to the drug.

Adrenalin or some other vasoconstrictor should always be a part of the retrobulbar injection. Otherwise, slight edema of the posterior orbit is likely to result.

Ballooning the bulbar conjunctiva by the injected novocaine indicates the retrobulbar injection was not in the muscle cone. In such an instance, a further injection of 1 cc. more of novocaine may be given, provided the eye is not unduly prominent.

3. A tight palpebral fissure should be remedied at the start of operation by a large, deep lateral canthotomy. A Parke lid speculum holds the lids open better than any other speculum we have found. Also, it does not slip out of place and the blades do not press on the globe. Some surgeons insist on lid sutures in lieu of a speculum, or on a speculum which can be removed very quickly in the event of vitreous loss. The logic of this contention is not clear. When corneoscleral sutures are used, the lids must certainly be held open until the incision has been properly closed. After the incision is closed, the speed with which the speculum is removed is of no consequence.

4. Lack of excessive retrobulbar bulk. If the eye is very prominent, or if there is excessive intraorbital resistance, a retrobulbar injection will cause a further increase in the orbital contents with increased pressure on the globe. In such patients general anesthesia is preferable.

Orbital hemorrhage from retrobulbar injection is very rare, but may occur if the needle is inserted too close to the globe and hits the inferior oblique muscle. Hemorrhage in the orbit, manifested by proptosis and by ecchymosis of the conjunctiva, is usually evident before the operation is started. In such cases operation should be postponed.

After section has been made, if there is increased orbital pressure, it is manifested by forward prolapse of the lens-iris diaphragm against the back of the cornea. Extreme pressure even causes bulging of the iris into the incision, and spells danger of vitreous loss. When this happens, the speculum blades and superior rectus suture should be readjusted to give as little pressure as possible. As the lens is tumbled and delivered, the cornea should be kept pressed against the back of the lens with an Arruga expressor, thus holding back the vitreous. The sutures should be tightened immediately following delivery, before repositing the iris.

If there is actual loss of vitreous and the lens is delivered with intact capsule, the sutures should be tightened as quickly as possible. The iris will then either be prolapsed into the incision or folded backward. If it is prolapsed, a complete iridectomy should be performed and the pillars reposited. If it is folded backward, nothing should be done to the iris; the final pupil will be displaced upward and have the appearance of an iris coloboma, but the folding back of the iris does not in itself cause irritation.

B. Rupture of lens capsule

Rupture of the capsule during delivery makes complete removal of the lens material more difficult. Retained lens material may necessitate subsequent discission or capsulectomy, procedures far from benign. Also, the chances of postoperative "uveitis" and/or glaucoma are considerably enhanced. Reten-

tion of much lens cortex is rare except when the capsule of an immature lens is ruptured. Fortunately this is a rare occurrence. The capsules most easily ruptured are those of densely white, swollen, mature, or slightly hypermature cataracts.

When the capsule ruptures, as it will in a variable percentage of cases, the method of extraction varies according to the position of the lens when the rupture occurs. If the lens has already tumbled, it may often still be delivered in toto by taking another larger grasp with the forceps. If it has not started tumbling, the nucleus should be expressed and as much as possible of the cortex removed by irrigation. Then, if the eye is "soft," the residual capsule can usually be removed, without appreciable danger of rupturing the anterior hyaloid membrane, by sliding Arruga forceps in with the faces of the blades *parallel* to the iris plane and grasping the capsular remnants (fig. 1). Removal of capsule is then often facilitated by using two Arruga forceps, taking successive bites in the capsule as it is pulled out through the incision. If there is any pressure on the eye, with apparent danger of vitreous loss, residual capsule had best be left alone. An iridectomy is not necessary.

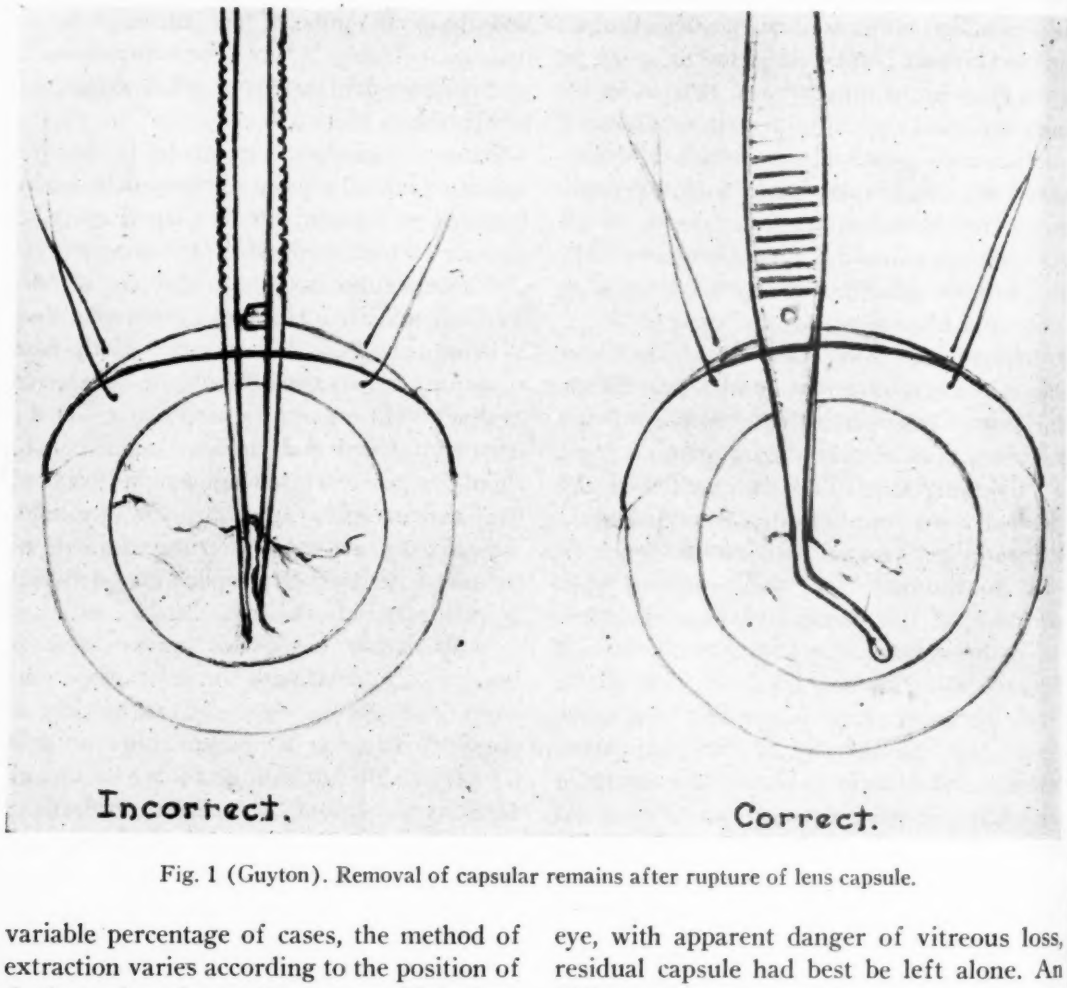

Fig. 1 (Guyton). Removal of capsular remains after rupture of lens capsule.

C. Vitreous loss plus rupture of capsule

If the lens capsule ruptures and vitreous begins to emerge at about the same time, the iridectomy usually should be completed. Lens material should then be removed by means of forceps and/or spoon, avoiding irrigation. The bulk of the lens material must be removed, but it is not advisable to

remove all lens capsule if such a maneuver can only be accomplished at the expense of much loss of vitreous. The corneoscleral sutures should then be tightened as quickly as possible, the iris pillars reposited, and judicious prayer indulged in.

D. Difficulties with section

Care should be taken immediately preceding section that the superior rectus suture has not been placed improperly and has not slipped.

With the assistant fixing the globe, it is quite rare that a suture is cut with a Graefe knife. If one or both sutures are inadvertently cut, the sutures are simply reinserted before the lens is delivered.

If the knife is tilted improperly, or if it has a defective blade which prevents it from cutting smoothly, aqueous may be lost before the counterpuncture is made and the point may become entangled with iris, back of cornea, or even lens. In this event, the knife should be removed, the chamber filled with air, and the section started over again. The same remedy should apply if a Graefe knife is carelessly inserted backward.

The anterior chamber may collapse after the counterpuncture but before the section is completed, with prolapse of iris in front of the knife (1) if the surgeon tilts the knife improperly and allows aqueous to escape too soon, or (2) if there is excessive orbital "pressure."

In the former instance, the iris can either be milked back over the edge of the knife or the chamber can be filled with saline or air with the knife in place. The section can then be completed without cutting the iris.

In the latter instance, a stream of saline directed along the knife may keep the iris out of the way or it may be necessary to remove the knife and complete the section with scissors. Stevens' scissors, inserted with the curve *downward,* are preferable for completing or enlarging a section. It is never necessary to go ahead and cut the iris with the knife.

E. Expulsive hemorrhage

An explusive hemorrhage during operation almost always results in loss of vision, with such disorganization of the globe that it should be enucleated as soon as permission can be obtained. Only extremely rarely can an eye be saved by immediately tying the corneoscleral sutures and evacuating the choroidal hemorrhage through a posterior sclerotomy several days later.

F. Partial dislocation of lens before it is grasped

Very occasionally the lens will partially dislocate at the time of the section, before the forceps are applied. This may be accompanied by spontaneous prolapse of vitreous into the anterior chamber. In this case it is best to perform a complete iridectomy and remove the lens with an erisophake and/or loop.

G. Complete posterior dislocation of lens

With extreme rarity an intact lens may dislocate completely and sink into the back of an eye. This occurs only if the vitreous is fluid. Such an eye is best closed without attempting to remove the lens. The lens can be removed later if the eye remains irritated. If removal is necessary, it may be possible to trap the lens in the anterior chamber, or it may be necessary to remove it by means of irrigation and a loop or a hook, or even by opening the eye and then turning the patient face downward while extracting the lens. If the lens capsule is ruptured, a posteriorly dislocated lens should be removed immediately.

H. Bleeding into the anterior chamber

Bleeding into the anterior chamber during operation generally occurs before delivery of the lens. The blood comes either from the incision or from a small iridodialysis. In either event, the bleeding should be allowed to stop completely and the blood removed from the anterior chamber before the

lens is delivered. Hemorrhage is rarely a result of inadvertently grasping iris with the intracapsular forceps and tearing the iris root, or from too forcible use of a spatula for reposition of the iris.

I. Incarceration of iris in incision

If the corneoscleral sutures are tightened before the iris is reposited, strands of iris are likely to be drawn into one or more of the suture tracts. The iris should be smoothed back into place before the sutures are actually tied. Care should be taken to free the iris strands with a spatula; otherwise the pupil will subsequently be "peaked."

J. Distribution of iris pigment

If considerable iris pigment is dislodged during an otherwise uneventful extraction, the anterior chamber should be irrigated gently.

Postoperative management

Eserine (¼-percent solution) is instilled once or twice, and a monocular dressing with shield is applied. If there has been rupture of the lens capsule, or if the patient has any chronic injection of conjunctiva or lid margins, an antibiotic is administered systemically as a prophylactic measure for several days. He is allowed out of bed the day of operation. Atropine (1-percent solution) is instilled the day following operation and the pupil is kept about medium size thereafter until the aqueous ray is negative.

The corneoscleral sutures are removed not earlier than the 11th postoperative day. After thoroughly anesthetizing the eye with instillations of 4-percent cocaine solution, the knot of each suture is cut with the sharp tip of iris scissors, with the scissors pointed upward, before the suture is grasped with forceps. If the patient has a tendency to squeeze the eye, removal of the sutures is delayed a week or longer. Induction of akinesia by means of an O'Brien block is occasionally advisable.

If vision in the other eye is very poor, a temporary lens is prescribed within two weeks after operation.

Postoperative complications

A. Infection, uveitis, lens sensitivity

These three complications are grouped together because a clear-cut distinction between them is not always possible.

A purulent infection will rarely develop around a corneoscleral suture. It is manifested by infiltration around the suture and is an indication for immediate removal of the suture, plus antibiotics. Failure to remove the suture promptly may lead to purulent endophthalmitis.

Purulent endophthalmitis may occur without relation to sutures, apparently from introduction of bacteria into the eye at the time of operation. Only a few years ago almost all eyes with postoperative purulent endophthalmitis were lost. The majority are now saved by the use of antibiotics.

The terms postoperative "uveitis" or "iritis" indicate merely a nonpurulent intraocular inflammation. Such inflammation may result either from undue traumatism, imperfect healing, or a very low-grade infection. Antibiotics are rarely beneficial for these nonpurulent inflammations, but foreign-protein therapy often appears to help.

Lens sensitivity may develop if there is retained lens cortex. There is considerable evidence that the development of sensitivity is dependent on a low-grade infection, but this is not altogether clear. Lens sensitivity does not develop until at least six days after operation. It is manifested by marked inflammation of the anterior segment, with gradual formation of a cyclitic membrane. The patient usually shows a positive skin reaction to 0.1 cc. of 2-percent lens protein injected intracutaneously. Such a skin test should be read 48 hours after injection.

If development of lens sensitivity is recognized early, the eye should be opened and the retained lens material removed as completely as possible with forceps, regardless of vitreous loss. In any event, the patient should

be desensitized. For this purpose, injections of 0.1 cc. of 2-percent lens protein plus staphylococcus toxin are given twice a week. The concentration of staphylococcus toxin employed depends on the patient's reactivity to test doses; a 1:100 dilution is most frequently used. Desensitization is carried out for at least six weeks prior to extracting a cataract from the other eye, as prophylaxis in case its capsule ruptures.

B. Glaucoma

Glaucoma following cataract extraction may result from postoperative uveitis or from extensive anterior synechias. Such synechias may result if the anterior chamber remains flat for a few days during the healing period. Collapse of the chamber is uncommon with the use of two corneoscleral sutures, but it very occasionally does occur, usually with an associated combined detachment, at any stage before the sutures are removed and especially at the time they are removed. If it remains collapsed for as long as three days, it should be reformed with air.

If postoperative glaucoma cannot be controlled with drugs, the operation of choice should usually be a cyclodialysis.

C. Anterior-chamber hemorrhage

A postoperative anterior-chamber hemorrhage usually results from rupture of newly formed capillaries in the healing incision. It may occur at any time within 2 or 3 weeks after operation, usually in 4 to 6 days. Such hemorrhages almost always clear completely, but some blood usually seeps into the vitreous and absorbs rather slowly. Blood should be evacuated from the anterior chamber only if it fills the chamber, has definitely clotted, and shows no appreciable absorption after 3 or 4 days.

D. Detachment of retina

A simple detachment of the retina is considerably more common in an aphakic than in a phakic eye. Hughes and Owens found it occurred 10 times as often after marked loss of vitreous as after no vitreous loss, but no more often following intracapsular extractions than following extracapsular extractions. The prognosis for reattachment of the retina in an aphakic eye is not as poor as is widely believed. Almost 30 percent of such retinas can be reattached with one diathermy operation, and almost 30 percent of those reoperated can be reattached with a second operation.[8]

E. Prolapse of iris

Iris prolapse is extremely rare if two properly placed corneo-scleral sutures are used. It is no more frequent with merely a peripheral iridotomy than with a full iridotomy. If an iris prolapse is small and is completely covered with conjunctiva, it need not be repaired. If it is large or not covered with conjunctiva, it should be excised as soon as it occurs and an additional corneoscleral suture inserted.

F. Capsular remains, occlusion of pupil, displacement of pupil

If the lens ruptures and there is retained capsule, the capsular remains may be dense enough to require a discission or capsulectomy as soon as absorption of lens cortex is obviously complete. Even more frequently, a retained posterior capsule which is initially transparent may in later years develop Elschnig bodies and require a discission.

Postoperative uveitis may result in an occluded or displaced pupil, or incarceration of iris or vitreous in the incision may cause the pupil to be grossly displaced. In such instances, a central pupillary opening may have to be made with a discission knife or with scissors.

G. Rupture of anterior hyaloid membrane

Over 50 percent of patients who have round-pupil intracapsular extractions develop a break in the anterior hyaloid membrane some months after operation. The incidence is lessened if the pupil is kept me-

dium-sized and mobile, instead of widely dilated, during convalescence. Usually the rupture causes no symptoms, but occasionally it produces transient signs of mild iritis and vitreous opacities. In such a case, the pupil should be kept dilated for a few weeks.

H. Miscellaneous

Gaping of the incision and/or filtering bleb is so rare with two corneoscleral sutures as to be of no real significance. We have never seen epithelization of the anterior chamber following an extraction with even one corneoscleral suture.

Striate keratitis rarely persists more than a few days after operation, but persistent striate keratitis and/or epithelial edema occasionally results if there was preëxisting endothelial dystrophy of the cornea, undue traumatism to the posterior surface of the cornea, or loss of vitreous. Epithelial edema may sometimes be partially alleviated by the use of a 5-percent sodium-chloride ointment.

Development of drug sensitivity, especially to atropine, is annoying but seldom of real importance; local use of one of the antihistamine compounds, such as 2-percent pyribenzamine ointment, is of value in controlling the reaction. Excessive astigmatism can result from improper coaptation of the incision. Sympathetic ophthalmia is extremely rare.

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References

1. Berens, C., and Bogart, D.: Certain postoperative complications of cataract operations. Tr. Sect. Ophth. A.M.A., 238-283, 1938.
2. Kirby, D. B.: Prevention and handling of complications arising during and after cataract extraction. Arch. Ophth., **25**:866-901, 1941.
3. Hughes, W. F., Jr., and Owens, W. C.: The extraction of senile cataract. A statistical comparison of various techniques and the importance of preoperative survey. Am. J. Ophth., **28**:40-49, 1945.
4. ———: Postoperative complications of cataract extractions. Arch. Ophth. **38**:577-595 (Nov.) 1947.
5. Lindner, K.: Ueber abaenderungen der intrakapsulaeren Staroperation. Ber. der Ophth. Gesellschaft., **52**:392-400, 1938.
6. McLean, J. M.: A new corneo-scleral suture. Arch. Ophth., **23**:554-559, 1940.
7. Stallard, H. B.: A corneo-scleral suture in cataract extraction. Brit. J. Ophth., **22**:269, 1938.
8. Grafton, E. G., Jr., and Guyton, J. S.: The value of injecting saline into the vitreous as an adjunct to diathermy operations for retinal detachment. Am. J. Ophth., **31**:299-303 (Mar.) 1948.

CENTRAL CONTROL OF INTRAOCULAR PRESSURE BY ACTIVE PRINCIPLES*

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In a previous publication[1] we submitted experimental evidence to indicate the presence of a diencephalic center or centers which exercise a regulating control of intraocular pressure. The nature of this regulation is the subject of the present paper.

It was noted in some of our preliminary investigations that spinal fluid appeared to exert a variable action upon intraocular pressure. Exposure of the animal either to light or to darkness was one of the environmental factors which determined the change. A cyclic variation of tension in man during day and night has been observed by Thiel,[2] Schoenberg,[3] and others. The tension was found to fall during the day and to rise at night. Duke-Elder[4] suggested as possible causes the effect of extraocular muscles, the pumping action of the ciliary muscle on the scleral spur, redistribution of blood, and change in its osmotic value.

Exposure of eyes to light has been shown to result in a fall of tension, whereas, darkness induced an increase. The mechanism for this effect has been ascribed to constriction and dilatation of capillaries.[4] On the basis of these, as well as our, observations, we decided to investigate the relationship of light and dark exposure to changes in intraocular pressure and to diencephalic centers.

MATERIALS AND METHODS

Rabbits and ducks were the subjects used in these experiments. The intraocular pressure was determined tonometrically. Two of the rabbits had congenital hydrophthalmus. All the other animals were normal and varied from 14 months to 2 years of age. The animals were exposed for 4 hours to bright daylight in addition to a 300-watt bulb placed 50 cm. from the cage. In producing dark adaptation, the animals were kept for 12 to 15 hours in total darkness. Volumes of 0.5 to 0.7 ml. of spinal fluid from rabbits and 3 to 6 ml. from people with light and dark adaptation were injected intravenously into rabbits. Intraocular pressure was determined at 10-minute intervals for 1 hour. In a small number of experiments, aqueous humor from rabbits' eyes was injected intravenously, and crude water extracts of the anterior and posterior lobes of rabbit pituitary gland was administered subcutaneously. These rabbits were exposed either to light or to darkness.

In order to determine the variations of tension to light and to darkness, 10 normal rabbits were placed in total darkness and then exposed to light on 26 occasions. The intraocular pressure was determined under similar conditions in two normal ducks and in two hydrophthalmic rabbits.

RESULTS

The intraocular pressure was increased in normal rabbits transferred from total darkness to bright light. In most instances, the increase consisted of 3 or more millimeters of mercury, which was considered a significant change.[5]

In normal ducks, the intraocular pressure decreased upon exposure to light from darkness. The decrease was of 3 or more millimeters mercury. The ducks reacted in a manner similar to man. A reverse reaction had taken place in rabbits (Table 1).

In rabbits with congenital hydrophthalmus, there were no significant changes in tension upon exposure to light. The tension became increased in 3 out of 16 experiments.

When spinal fluid was obtained from rab-

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TABLE 1

CHANGES IN INTRAOCULAR PRESSURE DUE TO LIGHT ADAPTATION*

Type of Animal Tested	Number of Animals	Number of Experiments	Range Tension in Darkness in mm. Hg	Range Tension in Light in mm. Hg	Difference in Tension between Dark and Light	
					Number of Experiments	Tension
Normal rabbits	10	52	10 to 28	19 to 31	41 11	+3 to 8 0 to +2
Hydrophthalmic rabbits	2	16	11 to 25	11 to 23	7 6 2 1	0 − 3 +10 + 3
Normal ducks	2	15	15 to 23	11 to 17	1 5 9	− 2 − 3 −4 to −7

* Conclusion: In adapting itself from darkness to light, the intraocular pressure rises in normal rabbits is unaffected to some extent in rabbits with congenital hydrophthalmus, and is decreased in normal ducks

bits exposed to bright light and the fluid was injected intravenously into other rabbits, the tension became elevated 3 or more millimeters of mercury in most of the animals. On the other hand, spinal fluid obtained from rabbits kept in total darkness produced a reduction of tension.

Spinal fluid from people exposed to bright

TABLE 2

EFFECT OF INTRAVENOUS INJECTION OF SPINAL FLUID UPON INTRAOCULAR PRESSURE OF RABBITS*

Nature and Source of Spinal Fluid	Number of Experiments	Range of Tension in mm. Hg Before Injection	Range of Tension After Injection of Spinal Fluid	Difference in Tension Before and After Injection of Spinal Fluid	
				Number of Experiments	Tension
From rabbits exposed to light	14	15 to 25	15 to 29	9 3 2	+3 to 7 +1 to 2 0
From people exposed to light	6	21 to 31	16 to 25	1 4 1	−3 −6 −8
From people exposed to darkness	14	15 to 24	19 to 29	1 2 6 3 2	+7 +5 +4 +3 +2
From rabbits exposed to darkness	22	18 to 29	16 to 24	2 9 7 2 2	−5 −4 −3 −2 0

* Conclusion: Spinal fluid contains active principles which increase or lower intraocular pressure. Exposure to light or darkness is a factor which determines an excess of one or the other of the active principles. Spinal fluid from people has a reverse effect to that from rabbits.

light reduced the intraocular pressure in rabbits. Fluid obtained from people kept in darkness induced an elevation of tension in rabbits (Table 2).

Crude water extracts of the anterior lobe of the pituitary gland were injected into rabbits subcutaneously. The anterior-lobe extract of animals kept either in darkness or exposed to light showed an increase in tension in most of the experiments. There was no selective action. On the other hand, extracts of the posterior lobe of rabbits exposed to light increased the tension while extract of animals kept in darkness decreased it (Table 3).

The aqueous humor was removed from a number of rabbits kept either in darkness or in bright light. The humor was injected intravenously into other rabbits. No significant changes in tension took place.

Discussion

The experiments outlined in this paper indicate that there is a secretion of two active principles, probably by the posterior lobe of the pituitary body. One of the principles serves to raise and the other to lower intraocular pressure. The principles are secreted into the spinal fluid, apparently in in such quantities as to produce a balanced action upon tension. The experiments also indicate the influence of light and darkness in inducing greater secretion of one or the other of the active principles.

TABLE 3

EFFECT UPON THE INTRAOCULAR PRESSURE OF CRUDE EXTRACTS OF ANTERIOR AND POSTERIOR LOBES OF THE PITUITARY BODY REMOVED FROM RABBITS EXPOSED EITHER TO LIGHT OR TO DARKNESS*

Nature of Crude Extract of Pituitary Body	Number of Experiments	Range in mm. Hg of Intraocular Pressure Before Subcutaneous Injection of Crude Extract	Range in mm. Hg of Intraocular Pressure After Injection of Crude Extract	Difference in Tension Before and After Injection	
				Number of Experiments	Tension
Anterior lobe of rabbits exposed to light	10	15 to 24	20 to 26	1 3 1 4 1	+7 +5 +4 +3 +1
Anterior lobe of rabbits exposed to darkness	8	18 to 27	22 to 29	1 3 2 2	+10 +5 +3 −2 to −4
Posterior lobe of rabbits exposed to light	12	17 to 25	20 to 28	1 1 4 3 1 1 1	+7 +6 +5 +3 +2 +1 0
Posterior lobe of rabbits exposed to darkness	14	18 to 27	15 to 25	1 2 3 3 3 2	−6 −5 −4 −3 −2 −1

* Conclusion: These experiments with crude extracts suggest that the posterior lobe of the pituitary body secretes active principles which alter intraocular pressure in response to exposure to light or darkness.

Based in part on experiments reported previously,[1] as well as on the work in this paper, and partly on investigations still to be published, the mechanism of action of the active principles upon intraocular pressure has been formulated.

The mechanism consists of secretion of active principles, probably by the posterior lobe of the pituitary body. It is not unlikely that other areas in the region of the posterior lobe may be concerned with production of these principles. The active principles are thrown into the spinal fluid of the third ventricle and affect the autonomic diencephalic centers. The stimuli travel in the diencephalon and along the autonomic fibers to the eyes,[1] inducing among other effects either a dilatation or constriction of capillaries depending on the predominance of the type of active principle. Other investigators had demonstrated repeatedly[6-8] that a rise in intraocular pressure is associated with dilatation of capillaries and dark adaptation, whereas, a fall in tension follows capillary constriction and light adaptation. Feigenbaum[9] and others showed that changes of intraocular pressure in response to exposure to dark or light are not related to pupillary motility. Others workers failed to find any relationship to accommodation.

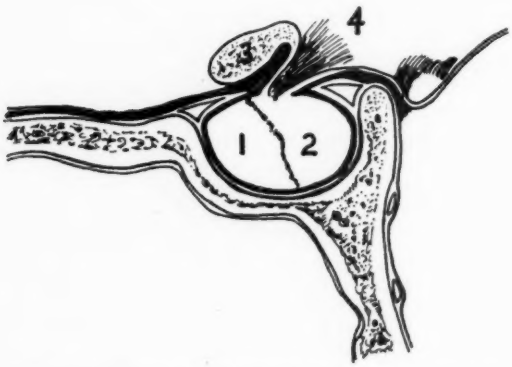

Fig. 1 (Schmerl and Steinberg). Anatomic relationship of the optic chiasma to the pituitary gland. (1) Anterior lobe of hypophysis. (2) Posterior lobe of hypophysis. (3) Optic chiasma. (4) Infundibulum. (After Gray's *Anatomy*, Philadelphia, Lea and Febiger.)

The mechanism of conduction of light and dark impulses from the retina to the posterior lobe may be explained on the basis of anatomic contiguity between the optic apparatus and the posterior lobe of the pituitary body (fig. 1) which, together with the tuber cinereum and the mammillary bodies, compose the hypothalamus.

Summary

Spinal fluid contains active principles, one of which increases and the other reduces intraocular pressure. Exposure of eyes to light or darkness is one of the factors which determines the quantitative secretion of one or the other of the active principles. On the basis of suggestive experiments, it is postulated that the active principles are secreted by the posterior lobe of the pituitary gland. These experiments indicate the probability that some forms of glaucoma may be induced by a disorder of the secretory mechanism of the active principles.

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References

1. Schmerl, E., and Steinberg, B.: The role of the diencephalon in regulating ocular tension. Am. J. Ophth., **31**:155-158 (Feb.) 1948.

2. Thiel, R.: Experimentelle und klinische Untersuchungen über den Einfluss des Adrenalins auf den Augendruck beim Glaukom. Arch. f. Augenh., **96**:331, 1925.

3. Schoenberg, M. J.: Experimental study of intraocular pressure and ocular drainage. J.A.M.A., **61**:1908, 1913.

4. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, C. V. Mosby Co., 1944, v. 1, pp. 502, 528.

5. Schmerl, E., and Steinberg, B.: Determination of ocular tension and rigidity in rabbits. Am. J. Ophth., **29**:1400, 1946.

6. Parsons, J. H.: The Ocular Circulation. London, 1903.

7. Wessely, K.: Experimentelle Untersuchungen über den Augendruck, sowie über qualitative und quantitative Beeinflussung des intraokularen Flüssigkeitswechsels. Arch. f. Augenh., 60:97, 1908.
8. ———: Ueber den Einfluss der Augenbewegungen auf den Augendruck. Arch. f. Augenh., 81: 102-119, 1916.
9. Feigenbaum, A.: Ueber den Einfluss der Belichtung und Verdunkelung auf den intraokularen Druck normaler und glaukomatöser Augen. Klin. Monats. f. Augenh., 80:577-596, 1928.

THE USE OF PLACENTAL EXTRACT IN THE TREATMENT OF HERPETIC KERATITIS AND SUPERFICIAL PUNCTATE KERATITIS*

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In therapeutics, the use of a homologous substance is rare. Most medications are plant extracts, minerals, or tissues of heterologous origin, rather than human tissue. An easily obtainable homologous tissue, one which contains a great number of important factors, is the human placenta. The placenta contains hormones, vitamins, enzymes, and antibodies in addition to many other factors. A tissue extract made from many pooled placentas contains a great variety of all these substances.

FILATOV'S WORK

Filatov[1] and his co-workers have reported the use of various types of tissues and tissue extracts. His work based on his theory that:

Tissues, which have been stored and treated under certain conditions, showed impaired viability, but these tissues became rich in substances which stimulate biochemical processes and regeneration. These substances have been called biogenic stimulators. The exact nature of these stimulators is unknown to us so far. These substances, when introduced into a diseased organism stimulate the regenerative processes of the organism and the pathologic processes diminish or disappear.

Filatov has reported the use of such extracts in a great variety of conditions. He has stated that they are especially valuable in such ocular diseases as retinitis pigmentosa, keratoconus, and optic atrophy.

The purpose of this paper is to make a preliminary report on the production and clinical uses of placental extracts. The work on which this paper is based has already occupied a year and a half, but it can be regarded only as ground work. Much remains to be done. To date we have been concerned with the following problems:

1. To break down the tissue extract into its component factors.
2. To determine, if possible, the nature of the biogenic stimulator.
3. To corroborate the work of Filatov.
4. To determine the effects of the use of tissue extracts on conditions not previously treated by this method.

PART I: PREPARATION OF PLACENTAL EXTRACTS

The technical method of the preparation of aqueous placental extract used in this investigation is essentially based on the method proposed by Filatov for the extraction of biogenic stimulators from homologous tissue. The biochemical work of making the various extracts was done by Dr. Edgar L. Milford of the Lederle Laboratories. An attempt was made to obtain uniform extracts in order better to evaluate the clinical results. The placental extract was first broken down into four factors:

* From the Eye Clinic of the New York Eye and Ear Infirmary. Read at the New York Society for Clinical Ophthalmology, May 5, 1947.

1. Aqueous extracts—containing water-soluble vitamins and hormones and most likely the biogenic stimulator.

2. Lipoid extract—containing cholesterin and lecithin.

3. Immune-gamma globulin—containing many antibodies found in human blood, and because of its pooled nature containing a greater variety.

4. Tissue coagulant—containing a factor which lowers the blood-clotting time.

Preparation of aqueous placental extract

Normal full-term placentas are transferred immediately after expulsion under aseptic precaution to sterile containers and placed in a freezer at 2°C. to 4°C. Only placentas from women with negative tests for syphilis and free from toxemia of pregnancy or any other disease transmissible by blood are used. The placentas are kept at 2°C. to 4°C. for two weeks prior to processing. After removal of blood clots and umbilical cord, the placentas are coarsely ground. The ground tissue is mixed with sterile distilled water in the proportion of 500 cc. per 450 gm. of placental mass. The mixture is stirred for one-half hour and then heated to 37°C., and the heat is maintained at that point for one hour. The mass is stirred constantly during the heating process. With the use of a hydraulic press at 70-tons pressure, the liquid portion is separated from the finely ground placental pulp and from coagulin formed during heating. The partly clarified extract is then sterilized by filtration through a 10-pound Mandel filter. This method of sterilization was judged less likely to effect the active principle than tyndallization in the event that the principle was thermolabile.

The stability of the extract being unknown, it was thought best to store the extract under conditions which have proved effective in minimizing the potency loss of other unstable biologic products. It was, therefore, concentrated into a dry powder, which was stored at 4°C. and used as the standard source of all therapeutic preparations in the investigation.

The aqueous extract found most satisfactory for clinical use was prepared by using 1 gm. of powder for 100 cc. buffered saline. The mixture was shaken and then dialized under aseptic precautions against three changes of saline. The protein content after sterile filtration was 4.9 percent. The extract was preserved by the addition of small quantities of phenol, which on comparative clinical studies was shown to have no effect on its potency. The clinical results obtained and reported in this paper are based on this dialized preparation.

It is interesting to note that the apparent effects of the extracts are consistently much greater when the placentas are first solidly frozen and stored for two to three weeks prior to processing than when the placentas are processed immediately after delivery. This seems to be in accord with Filatov's findings as to the importance of so-called unfavorable but not fatal conditions, such as storage at low temperature, in the development of biogenic stimulators.

The extract was also tested for its titers of immune antiviral and antitoxic bodies, before and after drying and heating. The overall diphtheria antiviral antibodies and influenza-A and B strains were less in the heated and vacuum-desiccated extract than in the original. Comparative fractional precipitation tests with ammonium sulfate showed that heating under the conditions described removed practically all the low beta globulins; whereas, gamma globulins and albumin were not affected.

It is also of interest to note that, despite prolonged dialysis against buffered saline, no appreciable loss of effect was found. This indicates that the active principle is probably in a colloidal condition or bound to a colloidal carrier.

Further attempts will be made to fractionate this standard extract with the view of recovering the active principle in some one fraction, which will be submitted to chemical

studies. The results obtained will be published at some later date.

Preparation of the lipoid extract

The lipoid extract was prepared in two fractions—ether and acetone soluble and acetone insoluble. The ether-acetone soluble fraction, which probably contains cholesterin, Factor A, was suspended in almond oil in 25-percent strength. This mixture was sterilized by passing it through a Sietz filter. The ether-acetone soluble fraction, found to contain mostly lecithin, was not used clinically because of the severe reactions it caused when injected intramuscularly in rabbits.

Preparation of immune globulin

This process will be described in detail in a paper to be published in the Journal.

To summarize briefly. Gamma globulins comprise about 11 percent of the plasma proteins and are found primarily in Fraction II, to a smaller extent in Fraction III.

Clinical interest in gamma globulin lies not in its physical constants, but in the fact that many of the antibodies elaborated by the defense mechanisms of the body are concentrated in this fraction.

Preparation of placental-tissue coagulant

The placental tissue coagulant is prepared essentially according to the method published by McKhann[2] in the *Journal of Pediatrics* for the treatment of hemophylia.

After placental tissues have been subjected to exhaustive saline extraction to obtain the immune globulin, the residue is extracted with cold phenolized saline at a temperature of 10°C. The pH of the mixture is adjusted to 9 with N/1NaOH, put through a homogenizer and allowed to stand in a cold room at 10°C. for 12 hours. The coarse placental tissue is separated from the extract with the use of a hydraulic press. The effluent is clarified by centrifugation through a Sharpless centrifuge. The clarified extract is precipitated at its isoelectric point (pH 5) by addition of N/5 hydrochloric acid, drop by drop, under constant mechanical stirring. The precipitate formed is allowed to settle in the cold room. It is suspended twice in acidified cold distilled water (pH 5.2). When the supernatant is colorless, the final precipitate is collected by centrifugation, then dissolved in weak sodium-hydroxide solution containing 0.5-percent NaCl The pH of the coagulant solution is adjusted to 7.5. This solution is centrifuged and sterilized by being passed through a medium Berkefeld filter.

The final coagulant solution in 1-cc. quantity will clot 1 cc. of reclarified horse plasma, with a control time of 10 minutes, in one minute. This test is used for determining the potency of the coagulant. The protein content of the final extract is about one percent.

Part II: Clinical report

This report presents the results of the work done with the various placental extracts in the treatment of a variety of ocular diseases. Twenty-four patients were treated at various times with some one or several of the extracts described in Part I of this paper. The results obtained indicate that the aqueous extract may be of value in the treatment of virus diseases of the cornea. Indications also exist that immune globulin may prove of value in the treatment of uveitis.

Aqueous placental extract in superficial punctate keratitis

Gratifying results were obtained with the use of aqueous extract in both superficial punctate keratitis and in herpetic corneal lesions. Six cases were treated in this group. It would seem to me most valid to assume that the presence of the biogenic stimulator in the aqueous extract gave impetus to the regenerative processes of the cornea. The assumption that the results were obtained because of the presence of antibodies in the placental extract seems to me untenable. The use of the concentrated gamma globulin failed to produce any improvement.

Four patients with superficial punctate

keratitis were treated with the aqueous placental extract described in Part I. The patients responded very favorably. Of the patients in this group, three had a permanent recovery after the injection of 3 to 5 full doses of the aqueous extract. They showed no recurrences upon subsequent examinations over a period of a year. These patients had previously failed to respond to local therapy, foreign-protein therapy, or administration of vitamins—all of which had been used over a period of one month. The third patient required more extended therapy but is now symptom free for as long as four months at a time.

The dosage of the aqueous extract depends upon the concentration. As of this writing, 2 cc. of concentrated solution is given intramuscularly.

Case reports

Case 1. Mrs. C. G., aged 45 years, consulted me first on April 25, 1946, complaining of recurrent attacks of pain of the right eye following an injury three months previously. A diagnosis of superficial punctate keratitis was made. The patient was treated locally until June 10, 1946, with some improvement but with frequent recurrences. She then received three full doses of the aqueous extract and by the 24th of June, the cornea no longer stained. Subsequent examinations did not show any staining nor reveal any recurrence.

Case 2. Mr. R. M., aged 42 years, consulted me on June 1, 1946, giving a history of recurrent attacks of pain, photophobia, and blurring of the vision of the left eye, of several months' duration. A diagnosis of superficial punctate keratitis was made. The patient received local therapy until June 17, 1946, without any improvement. Aqueous placental extract therapy was then instituted, and he received four full doses. By July 8, 1946, his lesions had healed and subsequent examinations revealed no other lesions.

Case 3. Miss R. B., aged 16 years, was referred from the service of Dr. Raymond E. Meek of the New York Eye and Ear Infirmary. The patient had first consulted clinic on April 26, 1946, giving a history of lesions of one year's duration. A diagnosis of bilateral superficial punctate keratitis was made. Prior to the injection of placental extract, she had received every local therapy known or suggested—foreign proteins, vitamins, iontophoresis, and thyroid extract because of her low basal metabolism. Her lesions disappeared for the first time after the injection of three full doses of the aqueous placental extract. The recovery in this case has not been permanent, but the patient has been symptom free for as long as four months. When she does have a recurrence she receives one or two injections of extract and again remains symptom free for several months.

Case 4. Miss M. M., aged 30 years, consulted me first on October 1, 1947, with a history of recurrent attacks of redness of both eyes lasting from 4 to 5 days and occurring 3 to 4 times a month. She had received various types of local therapy including a series of desensitization injections by another ophthalmologist for a period of one year. Two injections of 1 cc. of the aqueous placental extract healed the condition within 48 hours. Since this case is rather recent no definite conclusions can be drawn so far, but it does suggest that this therapy is effective in producing rapid healing of corneal lesions.

Aqueous placental extract in herpetic keratitis

In both of the cases treated for herpetic corneal lesions, the lesions were completely healed after several full doses of the concentrated extract. These patients had previously failed to respond to any local therapy including the use of ether. One of these cases was referred by another ophthalmologist specifically for treatment with this extract. The patient had failed to respond to a course of treatments which extended over a period of six months and included iodine application to the cornea and repeated curettage.

Case reports

Case 1. Mr. S. R., aged 70 years, consulted me on November 18, 1946, giving a history of local treatment for herpetic keratitis for 5 to 6 months. The treatment had been unsuccessful and he had had many recurrences. The first injection was given on November 14, 1946. On November 25, 1946, there was no longer any staining, and since then there has been no recurrence.

Case 2. Mr. M. A., aged 54 years, consulted me on March 23, 1946. A diagnosis of herpetic keratitis was made. Local treatment was given until September with constant recurrences. He was then given a series of placental-extract injections and, on September 24th, there were no staining areas. Two months later, this patient had a recurrence which was again treated with the extract. This cleared up shortly and to date there has been no further recurrences of the condition.

Other experiments with placental extracts

Aqueous placental extract was used in the treatment of retinitis pigmentosa. It cannot be said that the results obtained in this disease were as good as those reported by Filatov and his co-workers. They are, nevertheless, worth reviewing here.

Retinitis pigmentosa

Six cases of retinitis pigmentosa were treated over a period of one year. All received intramuscular injections of the aqueous placental extract and the lipoid extract.

Careful visual fields were done, and the visual acuity was recorded for each of the six patients prior to the use of these extracts. These examinations were repeated at frequent intervals during the course of the treatments. Whereas, Filatov reported increased visual acuity and enlargement of the visual fields as a result of his treatments, not one of the patients treated by me showed either one of these improvements at any time. What they did seem to show after injections of the extract was an apparent arrest of the deterioration. Although this is no criterion, since many patients continue for years without change, it has some significance. Two of the patients reported here had been under my care for at least a year prior to treatment with placental extracts and had shown exceedingly rapid deterioration of both visual fields and visual acuity. Examination showed an arrest of the disease upon treatment. These two patients, whose subjective evaluations are trustworthy, report also some improvement in night vision.

An attempt was made to treat all these patients with lipoid extract. The use of this extract was based on Filatov's findings that cod-liver oil injections were as effective as aqueous placental extract. If this is correct, it would indicate that the active principle would be of a lipoid nature rather than of a protein nature. The lipoid extract was prepared to test this assumption. However, the local reactions upon injection were so severe, that the use of the lipoid extract had to be discontinued until it could be refined.

Other conditions treated

In the hope that the presence of the biogenic stimulator might be of some benefit, the placental extracts were tried on four types of eye conditions whose treatment is unsatisfactory. Placental-extract injections were given in cases of old corneal opacities, tuberculous episcleritis, diabetic retinopathy, and macular edema as a result of angiospasm of a branch of the central retinal artery. None of the patients treated for these conditions showed any improvement or change in their condition as a result of the injections.

An important question to be answered is whether the effects obtained with the placental-extracts injections can be ascribed to the foreign protein in the extracts. The evidence would seem to contradict this possibility. Each of the patients who showed improvement upon injections of placental ex-

tracts, had previously received foreign-protein therapy without any improvement. It might be noted that the aqueous extract contains only 4.9-percent protein; whereas, foreign proteins are usually used in much higher concentrations. It is most likely, therefore, that the results obtained are due to some other factor, which is probably the biogenic stimulator.

Use of placental-tissue coagulant

One more placental extract at least warrants mention at this point—placental tissue coagulant, which lowers the blood clotting time. Its use has been demonstrated by McKhann in the treatment of hemophylia. Since hemorrhage is a well-known complication in surgery on diabetics, it was my theory that such an extract should be useful in this connection. I have used it prior to surgery, in two cases, with good results. The clotting time was lowered from 4½ to 1½ minutes in one case and from 3½ to 1½ minutes in the other. Both operations, a cyclodialysis and a cataract operation, were performed with a minimal amount of hemorrhage. Further use of placental-tissue coagulant has been temporarily suspended, however, until the side effects can be examined and studied in greater detail.

Summary

The preparation of aqueous placental extract, lipoid extract, and placental-tissue coagulant is described. A preliminary report on the use of aqueous placental extract in the treatment of herpetic keratitis and superficial punctate keratitis is given. The favorable results obtained in the treatment of these diseases indicate the need for further clinical investigation with a larger group of cases.

The favorable treatment of retinitis pigmentosa with placental extract, as reported by Filatov, was not corroborated by the results obtained.

Old corneal opacities, tuberculous episcleritis, diabetic retinopathy, and vascular changes in the retina, did not respond to treatment with placental extract.

Placental-tissue coagulant was used preoperatively to lower the blood-clotting time in diabetics.

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References

1. Filatov, et al.: Soviet-Am. J., 2:(no. 1, Oct.) 1944.
2. McKhann, C. F.: J. Pediat., 8:135, 1936.

END RESULTS IN RETINAL DETACHMENT

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The retina as a whole is not involved in retinal detachment, but a cleavage occurs between the two primitive retinal layers, with the pigment epithelium remaining in position attached to Bruch's membrane, while the inner retinal layers become separated from it.

The retinal pigment epithelium originates from the outer neurodermal layer of the embryonic optic cup. The remaining layers of the retina arise from the embryonic inner neurodermal layer. Thus, in retinal separation, the inner layer becomes displaced from the outer layer, allowing the potential space to become an actual space.

CLASSIFICATION OF DETACHMENTS

All retinal detachments may be classified as secondary and the cases of idiopathic detachment are probably caused by inflammatory or degenerative changes. Terry,[1] in examining microscopic sections of eyes, classified separated retina into four general types: (1) Retinal separation with typical hole in retina, arising from trauma. It may be associated with mild uveitis. (2) Retinal separation that arises from exudation under the retina. (3) Retinal separation with uveal melanomas. (4) Retinal separation with myopia.

INJURIES CAUSING DETACHMENT

The types of injuries causing retinal detachments are: (1) Direct injuries to the eye, including perforating wounds, intraocular foreign bodies, and contusions. (2) Indirect injuries to the eye, such as blows and contusions on the head.

Duke-Elder[2] states that the choroid is more easily ruptured than the healthy retina, and the normal eye can stand the most severe trauma involving even rupture of the sclera and the loss of vitreous without the occurrence of a detachment. We have seen a great many more tears of the choroid in war injuries than tears of the retina.

Severe inflammatory lesions have been recognized as etiologic factors in retinal detachment for a long time. It is only comparatively recently that a mild equatorial and anterior choroiditis has been considered as a precipitating factor in retinal detachment. In these cases, the detachment is due either to the formation of exudative subretinal fluid, to traction by fibrous tissue, or to shrinkage of the globe. In war injuries, we have seen severe vitreous hemorrhages which, by contracting fibrous bands, produce detachments as a late sequela.

INCIDENCE OF RETINAL HOLES

Gonin,[3] in his latest papers, obtained an 87-percent incidence of holes in retinal detachment, and it was his opinion that retinal holes were almost invariably found in retinal detachments, if sufficient time were spent in looking for them. Duke-Elder, in summing up the significance of retinal holes, states that the hole iself is not sufficient to cause the detachment. Other factors —disease, degeneration, and severe trauma —play a part in retinal detachment. The consensus of opinion today is that a retinal hole exists in nearly every case of retinal detachment except those caused by intraocular tumor. Retinal tears or holes may be single or multiple and of varied form and size.

TYPES OF RETINAL HOLES

The types of retinal holes usually seen in retinal detachment are:

1. Crescentic, horseshoe-shaped tears, the convexity is always turned toward the optic nerve and usually occurs in the upper half of the globe.

2. Round holes, occurring either singly or

in groups, are generally in the temporal half of the globe, usually in the supratemporal quadrant.

3. Irregular slits and fine sievelike perforations are usually seen near or at the periphery.

4. Dialysis or disinsertion at the ora serrata almost always occurs at the lower part of the globe—most frequently in the inferior and inferior temporal quadrants. Subsequent detachment spreads slowly backward and upward with delayed involvement of the macula and visual disturbance.

In war injuries, when the patient suffers severe, violent trauma, more than one hole is often found in retinal detachment. In several cases that we observed the retina was lacy, ripply, shrivelled, and sievelike, with many holes.

Retinal holes have been known to heal spontaneously by glial proliferation, as reported by Samuels.[4] However, the general tendency is for the hole to become larger and the retinal detachment to become progressive.

Mechanism of detachments

The mechanism of some of the detachments is fairly clear. This is true principally of those detachments due to a tumor pushing the retina inward; organization of fibrous tissue pulling the retina inward; tearing of the retina by sudden traumatic distension; wrinkling up of the retina in a shrunken globe.

Two other groups in which the mechanism is more or less clear are: (1) Exudative detachments which are not typically associated with a retinal hole, and (2) serous detachments in which a hole is characteristically present.

In spontaneous retinal detachments, the essential factor is the preceding inflammation or degeneration of the retina, vitreous, or choroid. In some of our traumatic cases, the retinal separation was secondary to gross ocular disorganization and was an incident in severe progressive ocular disease.

Clinical symptoms

The significant clinical symptoms of retinal detachment are:

1. The complaint of seeing "spots in the eyes," with the appearance of muscae volitantes, suggestive of degenerative changes in vitreous or peripheral choroiditis.

2. The symptom most commonly looked for is photopsia, the sensation of flashes of light, indicative of slight movements of the retina. The flashes of light are more significant when they are constantly projected from a single region of the retina.

3. If the detachment is large or central, vision will become blurred or completely lost. However, if the detachment is shallow and localized away from the macula, it may give rise to no symptoms and vision may be good. Usually the patient complains of a cloud or a curtain appearing in front of the eye.

4. An area of visual-field defect corresponding to the area of detachment is present. The red reflex is altered and appears gray over the detached area. As the eye is examined with an ophthalmoscope, it is noted that there is a change in the normal color of the fundus as the area of detachment is approached and that this area is out of focus with the rest of the fundus. The detached retina appears as a gray or bluish-gray veil with few or many folds; or it may assume the proportions of a large balloon and obscure the disc. The vessels seen rising angularly over into the area of detachment lose their normal light streak and appear as dark, almost black, tortuous cords.

All authorities agree that a retinal hole or tear is the rule. The tears, holes, or rents are characterized by their sharp borders and the visible red choroid. If no retinal hole is found, it may be because the hole is located too far peripherally or is hidden by a fold. Retinal holes usually do not occur in cases of choroidal tumor. Gifford,[5] however, in 1936, reported a case of choroidal tumor with a hole.

TABLE 1

SUMMARY OF 15 CASES OF RETINAL DETACHMENT

Patient (Name and Age)	Age of Detachment	Previous Surgery	Etiology	Holes	Vision Before Surgery	Vision After Surgery	Visual Fields
G.J. 19 yrs.	3 months	none	inflammatory	none	20/100 J-5	20/100 J-5	improved
R.B. 23 yrs.	36 months	none	degenerative	dialysis	H. M. J-0	20/100 J-4	improved
M.J. 35 yrs.	7 months	yes	trauma	yes	5/200 J-0	10/200 J-0	improved
B.J. 23 yrs.	5 months	none	inflammatory	none	5/200 J-0	5/200 J-0	improved
H.A. 32 yrs.	2 months	yes	trauma	dialysis	20/400 J-0	20/400 J-0	improved
R.Z. 23 yrs.	2 months	none	trauma I.F.B.	dialysis	L. P.	L. P.	improved
F.R. 24 yrs.	10 months	yes	blast	none	20/20 J-1	20/20 J-1	improved
R.H. 21 yrs.	11 months	none	inflammatory	yes	20/80 J-1	20/70–3 J-1	improved
F.L. 19 yrs.	3 months	yes	trauma	yes	L.P.	C. F. in temp. field	improved
C.O. 26 yrs.	5 months	none	trauma	dialysis	L.P.	10/400	improved
P.P. 24 yrs.	5 months	none	trauma I.F.B.	yes	L.P.	L.P.	improved
S.R. 29 yrs.	2 months	none	trauma	dialysis	Poor L.P.	sclerectomy clear L.P.	improved
J.E. 21 yrs.	26 months	yes	trauma I.F.B.	dialysis	L.P.	sclerectomy 20/100 J-5	improved
W.M. 25 yrs.	7 months	none	trauma I.F.B.	yes	10/200 J-0	enucleated	
E.J. 23 yrs.	6 days	2 I.F.B. removed	trauma	none	L.P.	enucleated	

LOCALIZING POSITION OF TEARS

The position of a retinal tear may be localized ophthalmoscopically by estimating the number of disc diameters 1.5 mm. from the tear to the disc or ora serrata. This is a gross approximate method and easily done. Each disc diameter is 1.5 mm. and the ora serrata is considered to be 8 mm. from the limbus of a globe measuring 24 mm. on its anteroposterior axis.

Drawing of the fundus with details and localizing the retinal hole in relation to retinal blood vessels is another helpful method.

Stine's method,[6] with tables for retinal localization, is a more accurate method. The patient uses a Schweigger perimeter at 19 cm. and looks at his own eye in a mirror, or uses a finger (propioceptive) if the macula is damaged. He looks at the tear by means of a giant scope and draws until he is beyond the range of the arc. The arc is rotated until it intercepts the ophthalmoscopic beam, the reading on the perimeter arm is noted, to-

gether with the angle through which it is turned. Tables for localization of retinal tear on the globe are then consulted.

Case reports

All of the patients with retinal detachments herein reported were operated on by different members of the eye staff of the Eye Center, Crile General Hospital.

The operative procedure consisted of making a large conjunctival flap, with good exposure of sclera. The area of detachment was outlined with methylene blue, and the scleral area so outlined was treated by means of a single-point, insulated-tip electrode which produced micropunctures. In many places the punctures were enlarged to insure adequate drainage of the subretinal fluid.

Drainage holes stay open longer, and the retina is more likely to fall back into position, when a special diathermy current (coagulation and cutting) is used. Partial drainage is thus established early in the operative procedure, and the retina is more easily caught by the adhesive choroiditis. Multiple diathermy punctures give adequate drainage; whereas, surface coagulation with two or three diathermy punctures may give inadequate subretinal-fluid drainage. In war injuries, where the retina often is shrivelled and has multiple holes, the entire affected area should be treated, in an effort to close all the retinal tears and cause adhesions of the retina around the holes.

Case 1

History. G. J., aged 19 years, was inducted into the Army on September 18, 1943. Each eye had normal vision, and there was no history of eye injury or disease. On March 15, 1945, the right eye became blurred. He was hospitalized on March 24, 1945, overseas. He was given bed rest and binocular bandadges were applied for three weeks. He was evacuated to Crile General Hospital on May 20, 1945.

Findings. Detachment of retina (inflammatory type), right eye, inferior temporal quadrant, with inactive chorioretinitis in the same area. No tear or disinsertion was found. In spite of conservative therapy, the detachment persisted. Vision was: O.D., 20/100, J5; O.S., 20/15, J1.

Operation. On July 20, 1945, a barrage was done between the 6- and 10-o'clock positions, and surface coagulation was applied between the 8- and 10-o'clock positions.

Result. October 20, 1945. The retina was flat. Vision was: O.D., 20/80, J5; and there was definite visual-field improvement.

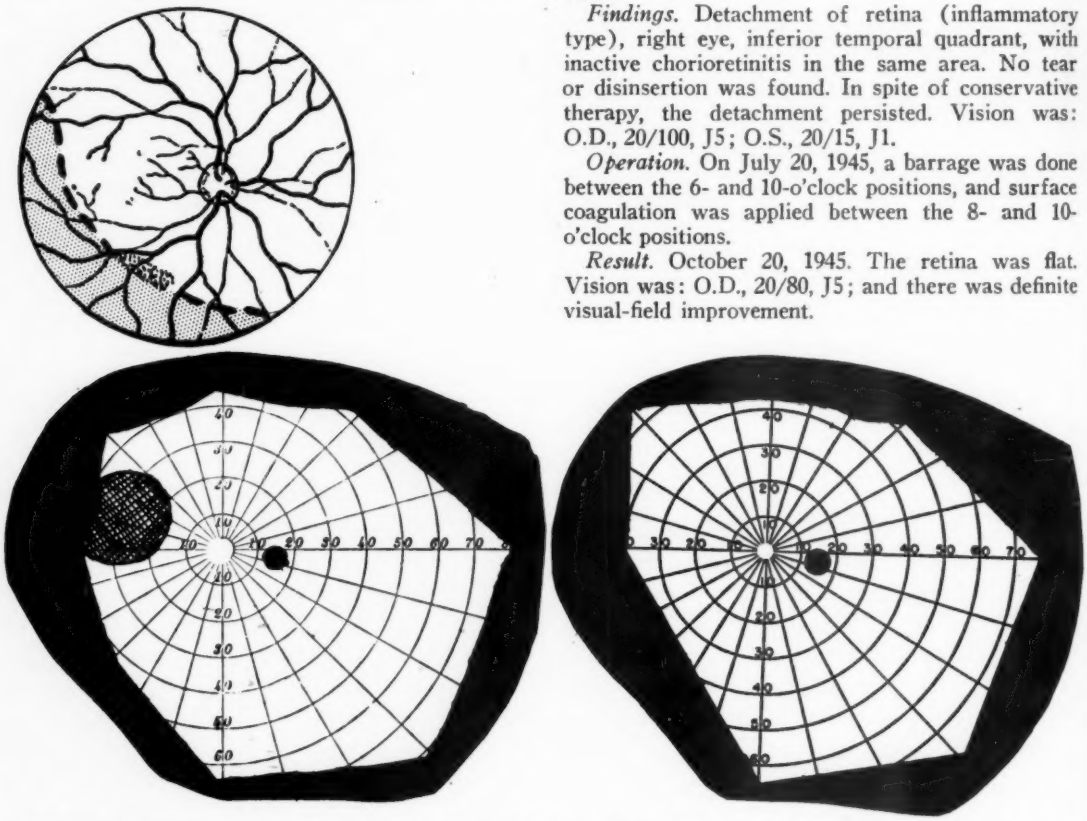


Fig. 1 (Croll and Croll). *Case 1.* Detachment of the retina (above), inferior temporal quadrant, right eye. Before surgery (left) vision was 20/100; fields for 3-mm. and 1-mm. white. After surgery (right) field for 3-mm. white; vision, 20/80.

CASE 2

History. R. B., aged 23 years, was rejected, in April, 1942, by the Navy for defective vision. He was inducted into the Army in September, 1942. In April, 1944, he tried to fire a carbine, sighting with the right eye, but he couldn't see with the right eye. He neglected to report this ocular condition until November, 1944. He returned from Iran on January 20, 1945.

Findings. There was a massive detachment in the right eye, with disinsertion of retina, involving the inferior temporal and nasal quadrant, right. Striae retinae were present above the macula. There was atrophy of the retina of the left eye in the inferior nasal quadrant, corresponding to the area of detachment in the right eye. Vision was: O.D., hand movements; O.S., 20/20, J1.

Operation. On March 20, 1945, an extensive barrage was done with a Liebel-Flarsheim single-insulated diathermy tip, from the 4- to the 9-o'clock positions. The power setting was 19.

Result. On July 17, 1945, the retinal disinsertion was still visible, but the retina adjacent to this was flat and adhered to the choroid. Vision was: O.D., 20/100 (marked improvement); definite visual-field improvement.

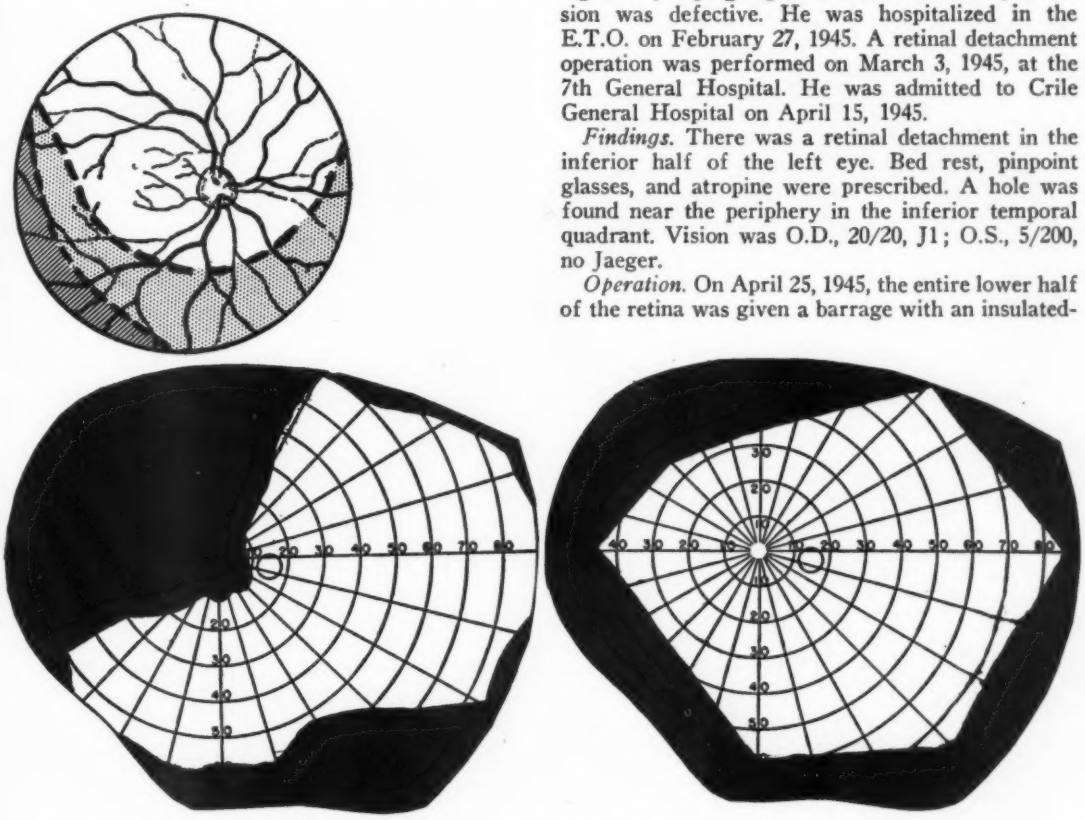


Fig. 2 (Croll and Croll). *Case 2.* Massive detachment (above) in the right eye, with disinsertion of the retina. Before surgery (left) vision was counting fingers at 2½ feet. After surgery (right) vision was 20/100 with correction. Visual field for 3-mm. white.

CASE 3

History. M. J., aged 35 years, during September, 1944, received a blow to the left eye from a fist. In December, 1944, the patient complained of flickering and jumping lights before the left eye. Vision was defective. He was hospitalized in the E.T.O. on February 27, 1945. A retinal detachment operation was performed on March 3, 1945, at the 7th General Hospital. He was admitted to Crile General Hospital on April 15, 1945.

Findings. There was a retinal detachment in the inferior half of the left eye. Bed rest, pinpoint glasses, and atropine were prescribed. A hole was found near the periphery in the inferior temporal quadrant. Vision was O.D., 20/20, J1; O.S., 5/200, no Jaeger.

Operation. On April 25, 1945, the entire lower half of the retina was given a barrage with an insulated-tip electrode. The retinal hole was sealed.

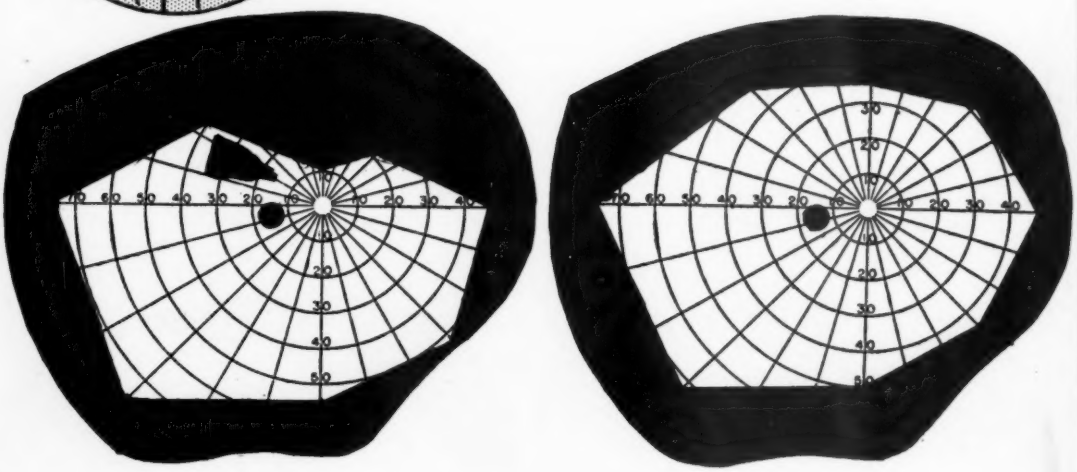
Result. On July 17, 1945, the retina was flat, and the adhesive choroiditis was holding well. Vision was: O.S., 10/200; with correction, 15/200. Visual fields showed definite improvement.

CASE 4

History. B. J., aged 23 years, had normal vision when he enlisted in September, 1940. There was no history of trauma. In November, 1944, the right eye became blurred and vision was only light perception. He arrived at Crile General Hospital on March 31, 1945, from the E.T.O.

Findings. There was detachment of the retina, right eye, with iridocyclitis and secondary glaucoma. No tear or disinsertion was found. There was increased intraocular pressure. A choroidal

Fig. 3 (Croll and Croll). *Case 3.* There was a retinal detachment (above) in the inferior half of the left eye. Before surgery (left) visual field for 3-mm. white; vision was 5/200; after surgery (right) vision was 10/200. Field for 3-mm. white.



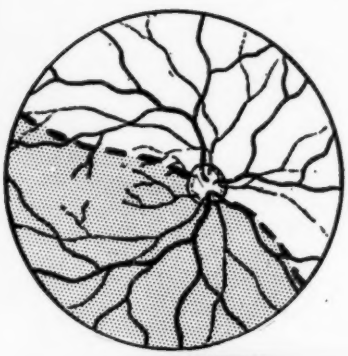
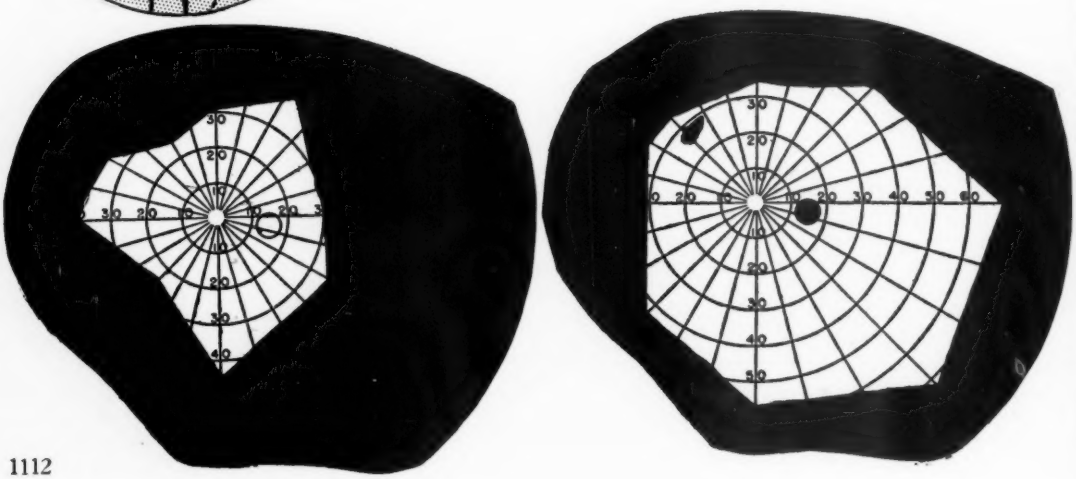


Fig. 4 (Croll and Croll). *Case 4.* Detachment of the retina (above) of the right eye, with iridocyclitis and secondary glaucoma. Vision before surgery (left) was 5/200; field for 3-mm. white. Vision after surgery (right) was 5/200; field for 3-mm. white.



tumor was considered. After treatment—paracentesis, atropine, foreign protein—a good red reflex was obtained by transillumination. Vision was: O.D., 5/200; O.S., 20/15, J1.

Operation. On April 27, 1945, an extensive barrage was done inferiorly between 3- and 9-o'clock positions with a Liebel-Flarsheim single, insulated diathermy tip. Power setting was 20, using a special retinal detachment current.

Result. On July 17, 1945, the retina was flat and the progressive detachment was cured. The eyeball was preserved. Vision was: O.D., 5/200, no Jaeger. There was marked improvement in the visual fields.

CASE 5

History. H. A., aged 32 years, was injured by fragments from mortar shell in France on February 2, 1945. On February 3, 1945, a multiple cautery procedure was done at an evacuation hospital. He was an ambulatory patient, then in bed for four days only, and again ambulatory. He was evacuated to Crile General Hospital, March 31, 1945.

Findings. In the right eye was a bullous, nearly complete retinal detachment, with retinal disinsertion between the 7- and 9-o'clock positions. There was a choroidal rupture through the macula. Vision was: O.D., 20/400, with head tilted to left; O.S., 20/15, J1.

Operation. On April 4, 1945, a large triangular laceration of the sclera was found at the 6-o'clock position, in front of the insertion of the inferior rectus muscle. It was sutured before diathermy punctures were applied over the sclera between the 5- and 10-o'clock positions.

Result. On July 15, 1945, the retina was flat and the progressive detachment was cured. The eyeball was preserved. Vision was: O.D., 20/200, with head tilted to left. There was marked improvement in the visual fields.

CASE 6

History. R. Z., aged 23 years, was wounded in action on October 18, 1944, in Italy. Mortar fragments penetrated his right eye. He had noted floating spots in right eye since injury. On July 27, 1945, a curtain came down over field of vision, from the temporal side of the right eye. He was admitted to the 17th General Hospital and was placed in bed for one month. Pinhole spectacles were prescribed. He was evacuated to the United States and arrived at Crile General Hospital on August 29, 1945.

Findings. An extensive retinal detachment was present in the lower half of the right eye. There was edema of the macula and a disinsertion from the 6:30- to the 8-o'clock positions. An intraocular foreign body less than 0.25 mm. in size was present in the inferior temporal anterior vitreous. It showed no motion with magnet application. There was slight

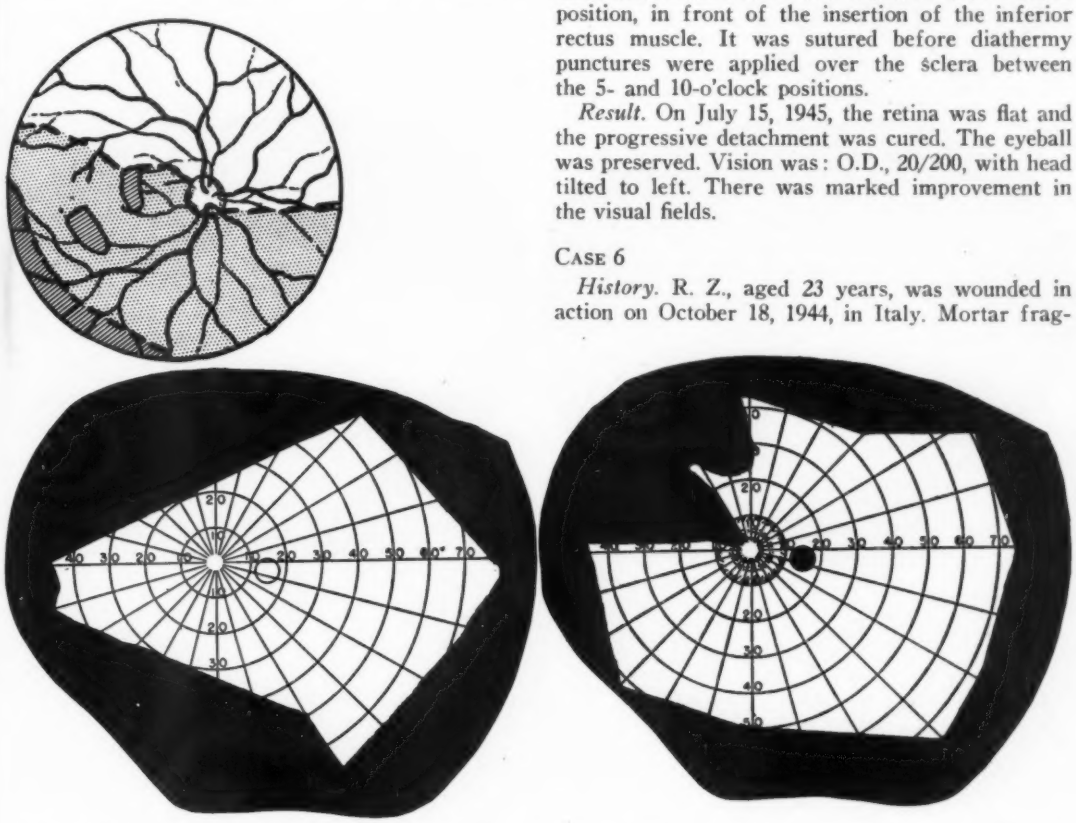


Fig. 5 (Croll and Croll). *Case 5.* In the right eye was a bullous, nearly complete retinal detachment. Before surgery (left) the target was an ophthalmoscope light and vision was light perception only. After surgery (right) vision was 20/200+ with correction. Target, 3-mm. white.

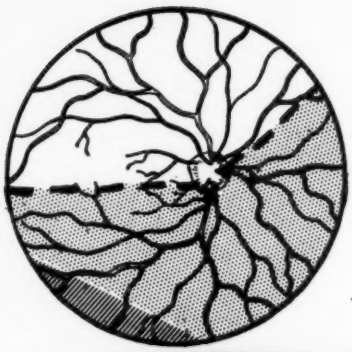


Fig. 6 (Croll and Croll). *Case 6.* An extensive retinal detachment (above) was present in the lower half of the right eye. Before surgery (left) visual field for 6-mm. white; vision was light perception. After surgery (right) field for 6-mm. white; vision was light perception.

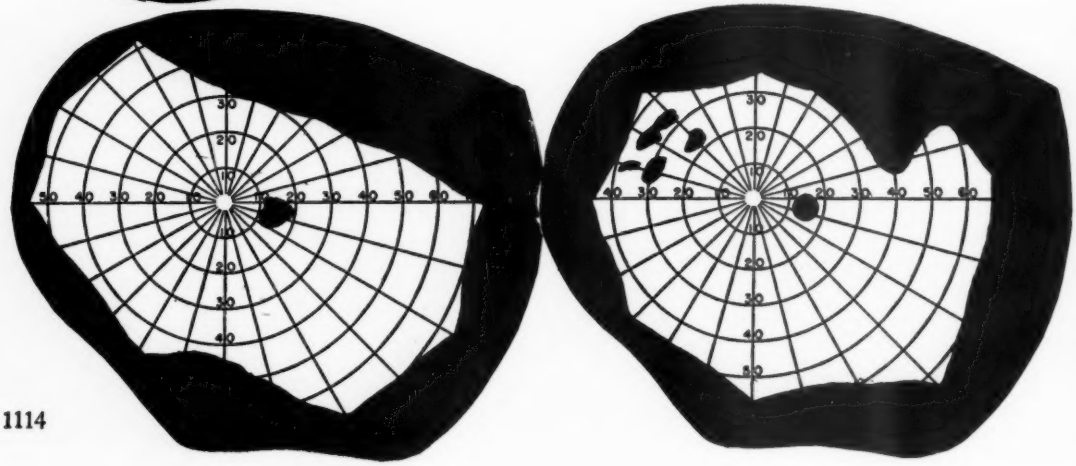


Fig. 7. (Croll and Croll). *Case 7.* The retina (above) was in position everywhere except at the extreme inferior nasal quadrant, right eye. Before surgery (left) vision was 20/100, correctible to 20/20; target for field was 3-mm. white. After surgery (right) vision was 20/100 correctible to 20/15. Field for 3-mm. white.

siderosis of the iris and lens capsule. Vision was: O.D., light perception; O.S., 20/20, J1.

Operation. One September 12, 1945, an unsuccessful attempt was made to remove the foreign body. An extensive barrage was done on the area of detachment, the inferior half nasally up to the 2-o'clock position.

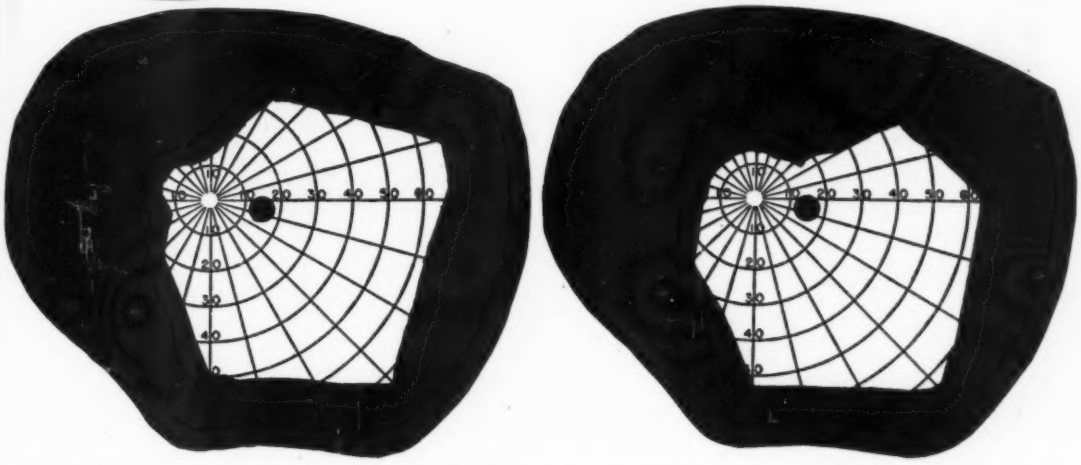


Fig. 8 (Croll and Croll). *Case 8.* While the patient was in bed, the retina, right eye (above), would flatten and the fields would become normal. When he was up for short periods, retinal detachment would recur. Before the second operation (left) vision was 20/80, correctible to 20/60. Target—3-mm. white. After surgery (right), vision was 20/80, correctible to 20/70+3. Target—3-mm. white.

Result. The retina was in good position, the extensive detachment cured and the eyeball preserved. Vision was light perception. The visual field showed improvement. The intraocular foreign body (nonmagnetic) was still retained.

CASE 7

History. F. R., was 24 years of age. While on Omaha Beach, one of our own demolitions exploded 20 yards from the patient. The blast was sufficient to throw patient over, even though he was lying down. On December 10, 1944, six months later, he first noticed a dark spot over the upper right eye. He was operated on March 1, 1945, at the 67th General Hospital and arrived at Crile General Hospital on April 4, 1945.

Findings. The retina was in position everywhere except at the extreme inferior nasal quadrant. No tear or disinsertion was seen. On April 24, 1945, the patient complained of flashes of light. There was definite deterioration of the field. The fundus showed a progressive detachment. Vision was O.D., 20/100, corrected to 20/20; O.S., 20/20.

Operation. On April 25, 1945, with a Liebel-Flarsheim single insulated diathermy tip, an extensive barrage was done from the 4:30- to the 9-o'clock positions. Power setting was 20.

Result. On July 18, 1945, the retina was flat, the progressive detachment cured, and a complete reattachment had been obtained. Vision remained 20/20. The visual fields were slightly improved.

CASE 8

History. R. H., aged 21 years, had normal vision in each eye in May, 1944. In August, 1944, the right eye became blurred, and he saw spots and flashes. There was no history of ocular injury. He was hospitalized in the E.T.O., and given bed rest, atropine, and pinpoint glasses for two weeks.

Findings. While the patient was in bed, the retina would flatten and the fields would become normal. When he was up for short periods, a retinal detachment of the right eye would recur. He was observed for several months. Before the first operation, no hole could be found, but after the first operation, a hole was found. Vision was: O.D., 20/80, corrected to 20/60; O.S., 20/20, J1.

Operation. On July 2, 1945, a surface coagulation of the sclera, from the 11- to 6-o'clock positions, was done. The sclera was light brown on completion. The detachment increased because there was insufficient drainage from two diathermy punctures, and only one half of the retinal hole was treated. On

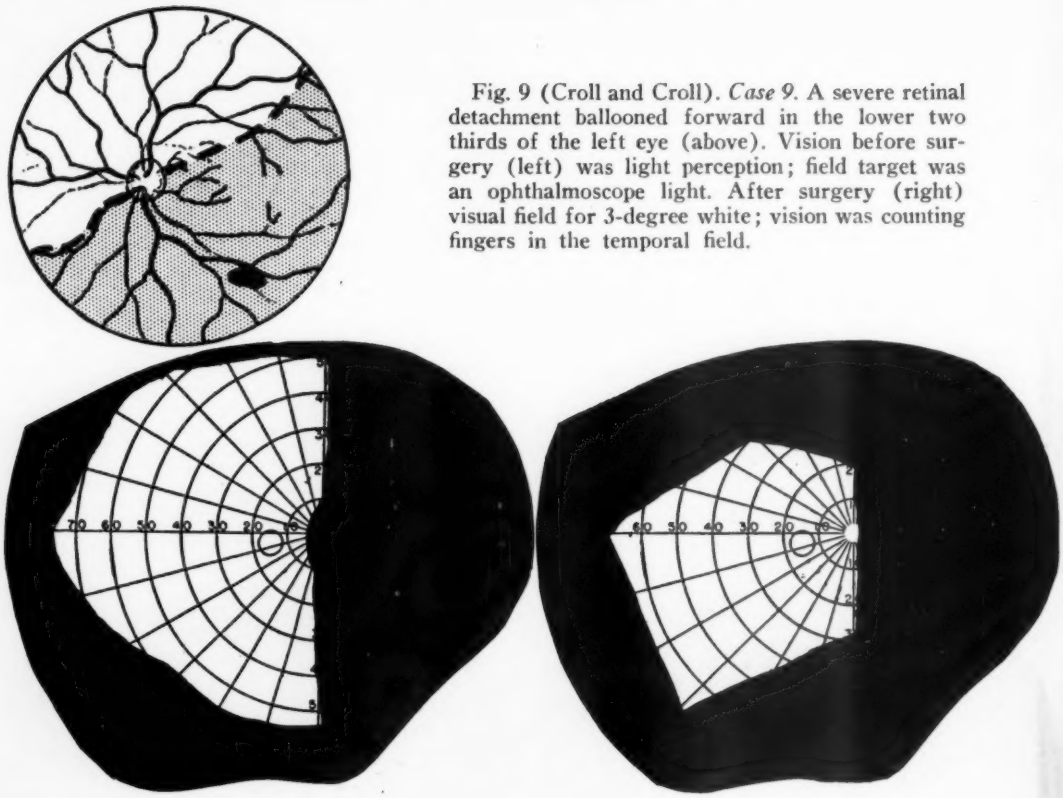


Fig. 9 (Croll and Croll). *Case 9.* A severe retinal detachment ballooned forward in the lower two thirds of the left eye (above). Vision before surgery (left) was light perception; field target was an ophthalmoscope light. After surgery (right) visual field for 3-degree white; vision was counting fingers in the temporal field.

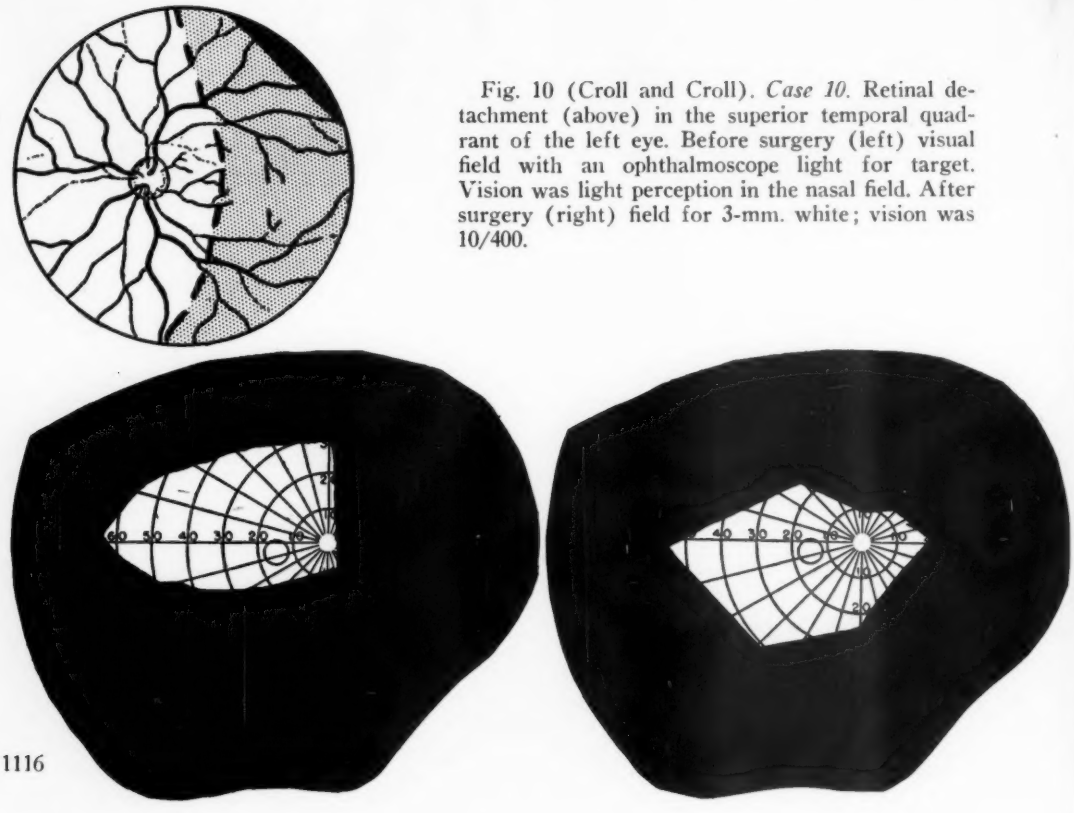
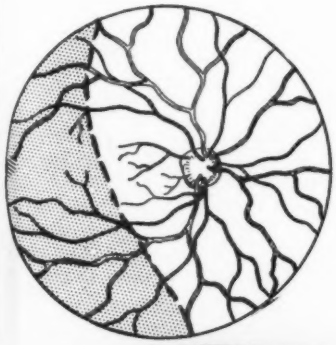


Fig. 10 (Croll and Croll). *Case 10.* Retinal detachment (above) in the superior temporal quadrant of the left eye. Before surgery (left) visual field with an ophthalmoscope light for target. Vision was light perception in the nasal field. After surgery (right) field for 3-mm. white; vision was 10/400.

July 25, 1945, the operation was repeated, an extensive barrage being used. The retinal hole was sealed.

Result. On September 20, 1945, the retina was in good position and completely reattached. Vision was: O.D., 20/80, corrected to 20/70+3. There was a slight contraction of the visual fields.



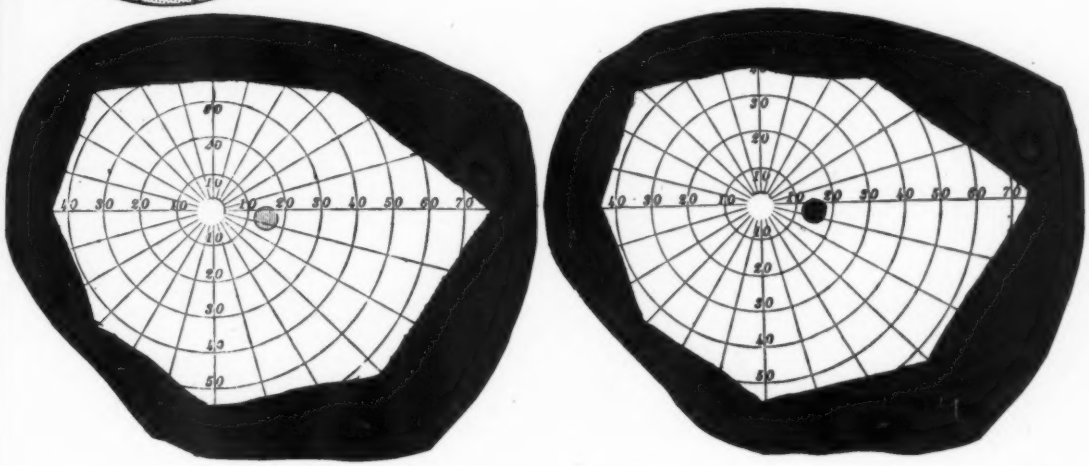


Fig. 11 (Croll and Croll). *Case 11.* Retinal detachment (above) of the right eye. Before surgery (left) vision was 20/30; field for 3-mm. white. After surgery (right) vision was 20/30; field for 3-mm. white.

CASE 9

History. F. L., was 19 years of age. On April 23, 1945, his left eye was injured by a fist in Germany. He was given bed rest for three weeks. When he was allowed up, the detachment of the retina recurred. On May 25, 1945, an operation for retinal detachment was done at the 15th General Hospital, Belgium, and the patient said that vision was poor following surgery.

Findings. A severe retinal detachment ballooned forward in the lower two thirds of the left eye. A retinal tear was present, inferiorly, at the 5-o'clock meridan, near the periphery. Vision was: O.D., 20/15, J1; O.S., light perception.

Operation. On July 5, 1945, a barrage of diathermy punctures was made from the 9- to the 3-o'clock positions. The area between the 4:30- and 6:30-o'clock positions was also treated with surface coagulation.

Result. On September 20, 1945, the retina was flat and completely reattached. The eyeball was preserved. Vision was: O.S., counted fingers in the temporal field. There was a slight contraction of the visual field.

CASE 10

History. C. O., aged 26 years, was wounded in action when a mortar shell exploded close to him on July 23, 1944.

Findings. There was a retinal detachment in the superior temporal quadrant of the left eye, with disinsertion from the 1- to the 3-o'clock positions. The macula was destroyed. Vision was O.S. poor light perception in the nasal field; O.D., 20/20, J1.

Operation. On January 10, 1945, a surface coagulation was done, but the detachment recurred. On April 9, 1945, an extensive diathermy barrage was done over the area of detachment.

Result. The retina was flat and completely reattached, and the eyeball preserved. Vision was: O.S., 10/400, with a slight improvement in the visual field.

CASE 11

History. P. P., aged 24 years, was wounded, March 8, 1945, in action in Germany by a fragment from a booby trap. A fragment was removed from the right eye at the Battalion Aid Station, and the sclera was closed with two silk sutures. At the 187th General Hospital, a diagnosis of retinal detachment was made. He arrived at Crile General Hospital on August 6, 1945.

Findings. A retinal detachment of the right eye was found in the extreme temporal quadrant, and there were vertical folds from the 7- to 11:30-

o'clock positions. Macular edema was present. A retinal tear was found at the 9-o'clock position in the far periphery. The vitreous contained about five small foreign bodies, which showed no movement on magnet application. X-ray films were negative for foreign bodies. Vision was: O.D., 20/30, J1; O.S., 20/20, J1.

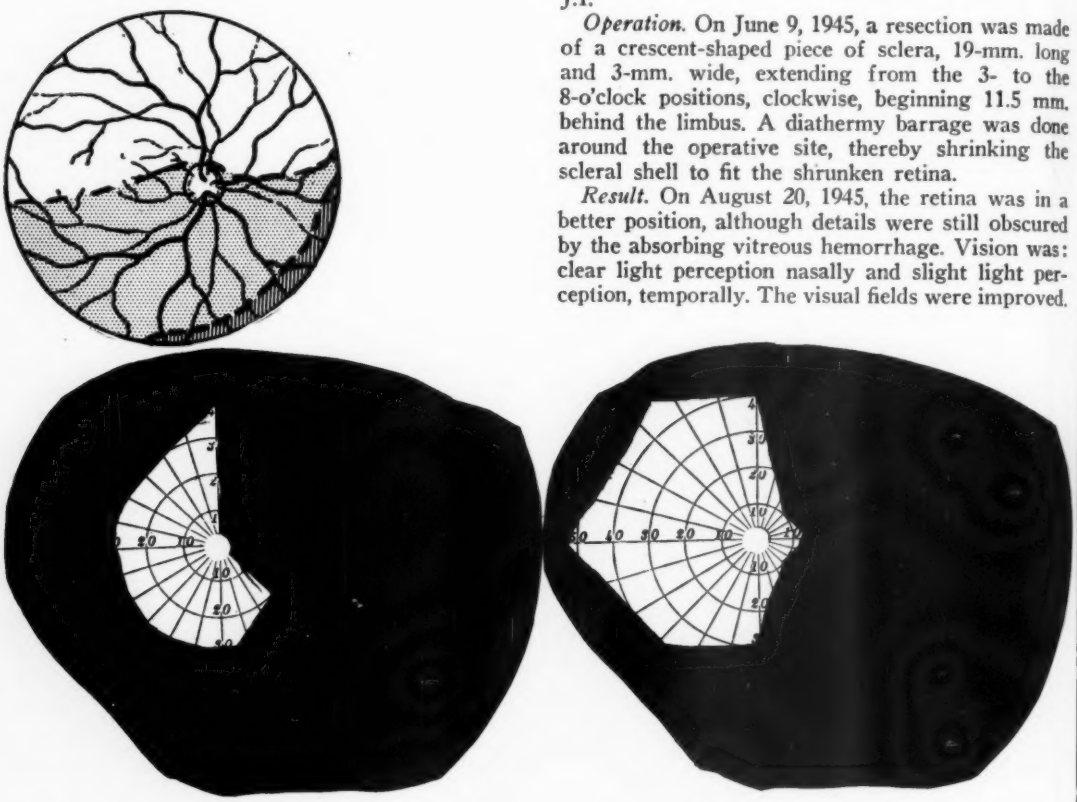


Fig. 12 (Croll and Croll). *Case 12.* Retinal detachment (above) in the inferior nasal-temporal area of the right eye. Before surgery (left) vision was light perception in the nasal field: target, ophthalmoscope light. After surgery (right) vision was clear light perception in the nasal field and slight light perception in the temporal field; target, ophthalmoscope light.

Operation. On August 14, 1945, a barrage was done from the 6:30- to 11:30-o'clock positions and the area from the 8:30- to 11-o'clock positions was treated by surface cautery, with a ball electrode over the area of the retinal tear.

Result. The retina was in good position and completely reattached. Vision was: O.D., 20/30, J1. The visual fields were unchanged. The nonmagnetic foreign bodies were still retained.

CASE 12

History. S. R., was 29 years of age. On April 4, 1945, in England, a grenade exploded close to him. He immediately lost nearly all vision in the right eye. Bed rest, pinhole glasses, and eye drops were prescribed.

Findings. There was a retinal detachment in the inferior nasal temporal area of the right eye, with a disinsertion between the 3- and 7-o'clock positions. An observer looking at the inferior aspect of the retina could see a shrunken retina with massive vitreous hemorrhage. Vision was: O.D., poor light perception in the nasal field only; O.S., 20/20, J.I.

Operation. On June 9, 1945, a resection was made of a crescent-shaped piece of sclera, 19-mm. long and 3-mm. wide, extending from the 3- to the 8-o'clock positions, clockwise, beginning 11.5 mm. behind the limbus. A diathermy barrage was done around the operative site, thereby shrinking the scleral shell to fit the shrunken retina.

Result. On August 20, 1945, the retina was in a better position, although details were still obscured by the absorbing vitreous hemorrhage. Vision was: clear light perception nasally and slight light perception, temporally. The visual fields were improved. On August 27, 1945, muscle surgery was performed for divergent strabismus, and anterior and posterior uveitis developed. On September 13, 1945, the right eye was enucleated to avoid possible sympathetic ophthalmia. It was feared that the scleral resection area had broken open, but on removal of eye, the area was found to be well healed.

CASE 13

History. J. E. was 24 years of age. On April 16, 1943, a bullet fragment ricocheted into his right eye. He was hospitalized at Walter Reed Hospital where an unsuccessful attempt was made to remove an intraocular foreign body. On April 1, 1945, while in Germany, he noted defective vision in the right eye. On May 1, 1945, he was operated on for retinal

detachment at the 52nd General Hospital, and another unsuccessful attempt to remove the foreign body was made.

Findings. A retinal detachment was present in the right eye. There were two large bullous areas above with a larger bullous area, lateral, and disinsertion from the 6- to the 10-o'clock positions. The retina was shrunken. Vision was: O.D., light perception only; O.S., 20/60, J1, correctible to 20/20, J1.

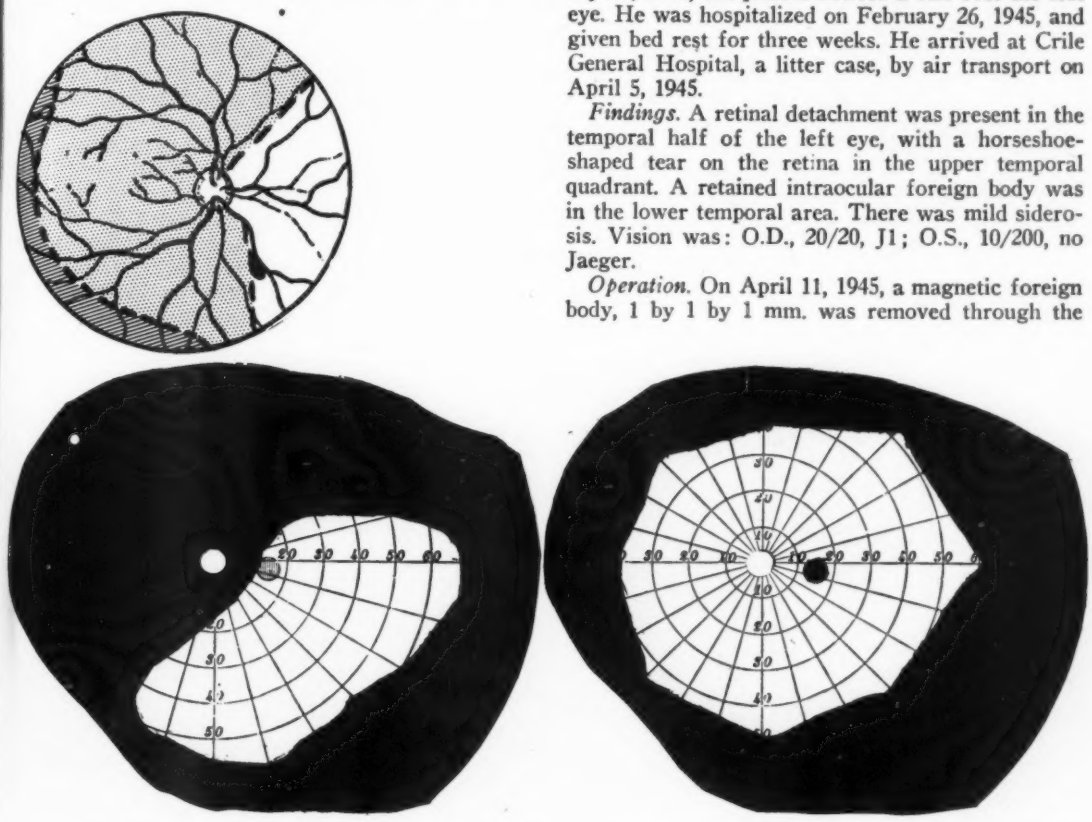


Fig. 13 (Croll and Croll). *Case 13.* Retinal detachment (above) of the right eye. Before surgery (left) vision was light perception; the target, ophthalmoscope light. After surgery (right) vision was 20/100; target, 3-mm. white.

Operation. On June 28, 1945, an eyeball shortening procedure of Müller[7] was done, with excision of a crescent-shaped piece of sclera, 20-mm. long and 2.5-mm. wide, extending from the 5- to the 10:30-o'clock positions, beginning 11.5 mm. behind the limbus. A diathermy barrage was done around the operative site. A fundus examination, immediately after surgery, showed the retina to be flat.

Result. On October 20, 1945, the retina was flat and completely reattached. The progressive detachment was cured. Vision was O.D., 20/100. There was definite visual-field improvement. The patient still had a retained foreign body, probably from the lead bullet (non-magnetic?).

CASE 14

History. W. M., aged 25 years, in September, 1944, was fixing a tire in Finchaven, New Guinea, when something struck his left eye. About February 25, 1945, the patient noticed a veil over his left eye. He was hospitalized on February 26, 1945, and given bed rest for three weeks. He arrived at Crile General Hospital, a litter case, by air transport on April 5, 1945.

Findings. A retinal detachment was present in the temporal half of the left eye, with a horseshoe-shaped tear on the retina in the upper temporal quadrant. A retained intraocular foreign body was in the lower temporal area. There was mild siderosis. Vision was: O.D., 20/20, J1; O.S., 10/200, no Jaeger.

Operation. On April 11, 1945, a magnetic foreign body, 1 by 1 by 1 mm. was removed through the pars-plana approach; then an extensive barrage was done with a single, insulated diathermy tip from the 6-to 12-o'clock positions.

Result. Plastic cyclitis developed, with posterior synechias. The anterior chamber was loaded with cells; fundus details were obscured. The patient did not respond to typhoid, penicillin, or atropine therapy. On May 8, 1945, the left eye was enucleated. There was no sympathogenic involvement of the right eye. Microscopic slides of left eye showed cyclitis with a cyclitic membrane, and detachment and gliosis of retina.

CASE 15

History. E. J., aged 23 years, was injured by a detonator-cap explosion in Germany on April 24, 1945. On April 30, 1945, he was operated on at the 203rd General Hospital and a foreign body was

removed. He arrived at Crile General Hospital on May 25, 1945.

Findings. There was poor light reflex and a massive vitreous hemorrhage of the left eye. Two intraocular foreign bodies were removed by the pars-plana approach, one temporal and one nasal, on June 11, 1945. The vitreous cleared. On August 23, 1945, the patient helped to lift a piano and a curtain closed over his left eye. Field and fundus examinations the next morning showed an almost total retinal detachment. Vision was: O.S., light perception only; O.D., 20/20, J1.

Operation. On August 29, 1945, after five days' rest in bed, an extensive barrage was done over the temporal half of the retina.

Result. The eye did poorly, and was enucleated on October 2, 1945.

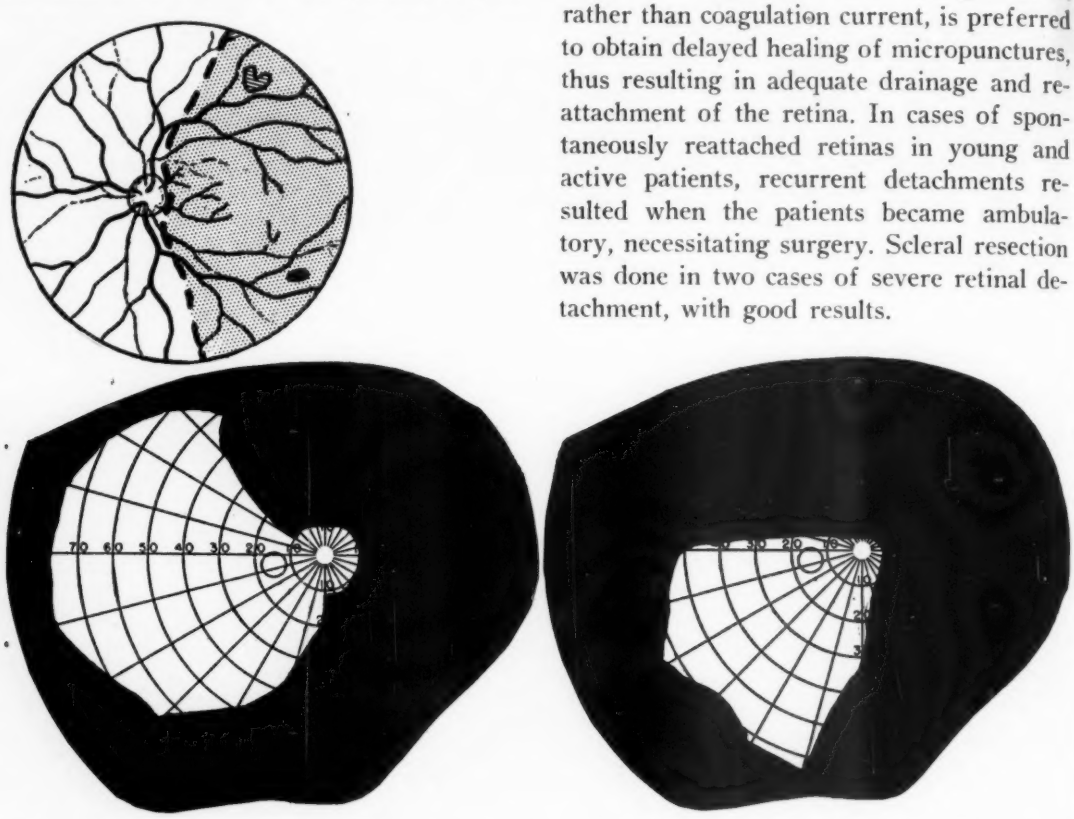


Fig. 14 (Croll and Croll). *Case 14.* A retinal detachment (above) was present in the temporal half of the left eye, with a horseshoe-shaped tear on the retina. Before surgery (left) field for 5-mm. white; vision, 10/200, not correctible. After surgery (right) field for 6-mm. white; vision, light perception only.

Comment

Areas of dialysis were present in six cases and peripheral holes in five, and no hole was seen in four cases. Extensive barrage was done over the affected area because of the severity of the injuries. The use of a combination of coagulation and cutting current, rather than coagulation current, is preferred to obtain delayed healing of micropunctures, thus resulting in adequate drainage and reattachment of the retina. In cases of spontaneously reattached retinas in young and active patients, recurrent detachments resulted when the patients became ambulatory, necessitating surgery. Scleral resection was done in two cases of severe retinal detachment, with good results.

Fig. 15 (Croll and Croll). *Case 15.* Poor light reflex and massive vitreous hemorrhage of the left eye.

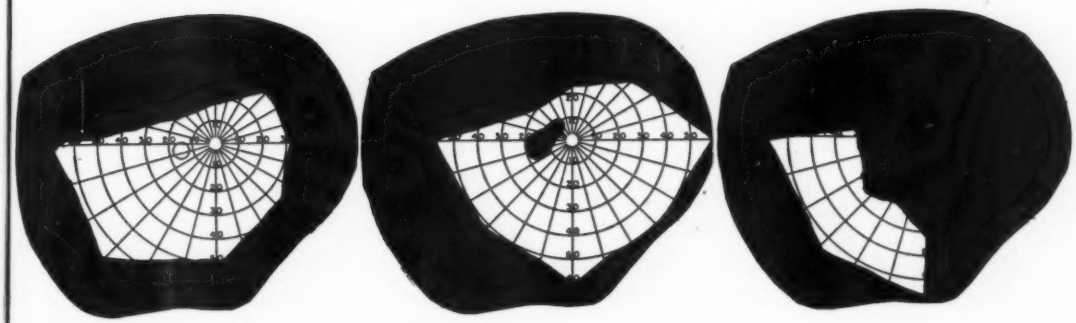


Fig. 16 (Croll and Croll). *Case 15.* Before surgery (left) visual-field target was ophthalmoscope light. (Center) Field for 3-mm. white after removal of two intraocular foreign bodies; vision, 20/400. (Right) Sudden and almost complete detachment after lifting piano. Target used was 18-mm.

Acknowledgement is made to Lieut. Col. G. C. Struble, Maj. H. G. Scheie, Maj. J. D. Sleight, and Captain Hoyt for allowing us to present these cases and to Sgt. Helen Hellebo who performed the visual-field studies and prepared the enclosed field records.

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References

1. Terry, T. L.: Tr. Am. Acad. Ophth., May-June, 1945, p. 272.
2. Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, v. 3, p. 2871.
3. Gonin, J.: Arch. Ophth., **4**:621, 1930.
4. Samuels, Bernard: Arch. Ophth., **21**:273-314 (Feb.) 1939.
5. Gifford, S. R.: Surgical treatment of retinal detachment. Arch. Ophth., **16**:405, 1936.
6. Stine, George H.: Am. J. Ophth., **17**:314 (Apr.) 1934.
7. Müller, L.: Eine neue operative Behandlung der Netzhautabhebung. Klin. Monatsbl. f. Augenh. **41**:459-462, 1903.
8. Pischel, D. K., and Miller, M.: Retinal detachment cured by eyeball shortening operation. Arch. Ophth., **22**:974-979 (Dec.), 1939.

THE FUNCTIONAL ORGANIZATION OF THE THIRD-NERVE NUCLEUS IN THE CAT*

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In 1942, Szentagothai showed by means of a modified Horsley-Clarke stereotaxic instrument, and a technique of direct stimulation of the oculomotor nerve nucleus that the representation of the striated ocular muscles within this nucleus for the cat and the dog is rather different from the one usually admitted.

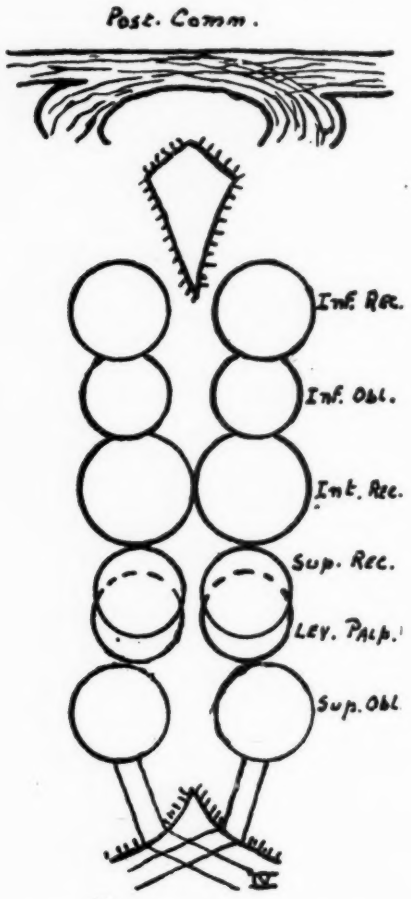

Fig. 1 (Danis). Functional organization of the third-nerve nucleus of the cat and the dog according to Szentagothai.

In 1943, Bender and Weinstein repeated this type of experiment on the monkey (Macaca Mulatta). Their conclusions were similar with those of Szentagothai.

According to these authors, the distribution of the radicular cells corresponding to the different eye muscles would be on the following pattern: going from the rostral to the caudal end of the third-nerve nucleus one would have the radicular cells for the inferior rectus, the inferior oblique, the internal rectus, and the superior rectus. The cells connected with the levator palpebrae would be grouped at the posterior end of the third-nerve nucleus against the fourth-nerve nucleus (fig. 1).

Szentagothai was unable to find any functional organization of the nucleus in a dorso-ventral direction. Bender and Weinstein believe that the different cell groups have more or less the shape of commas whose tails come under the head of the next anterior group. This arrangement would explain the fact that stimulation at different depths on the same dorsoventral axis gave different ocular movements.

None of these authors was able to show an organization of the nucleus in the transverse plane.

This type of experiment does not allow one to determine with certainty whether the innervation of the ocular muscles is crossed, direct, or mixed; it shows, however, that the majority of cells innervates the ipsilateral muscles.

These researches give a scheme of the functional organization of the third-nerve nucleus that is very different from those commonly published in ophthalmic and neurologic textbooks. The scheme more often

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reproduced for man is the one of Brouwer. This scheme is partly the extension to man of the experiments of Bernheimer on the monkey.

Bernheimer and all the other authors who have studied the localization of the eye muscles within the third-nerve nucleus have used the technique of retrograde degeneration (rabbit: van Biervliet, Bernheimer, Bach, Schwabe; cat: Abd-el-Malek Shafik, Latumeten). Szentagothai has also extirpated isolated eye muscles. The distribution of the retrograde degeneration in the third-nerve nucleus confirmed his results obtained by localized electrical stimulation.

The technique of retrograde degeneration is not in this case very reliable because the tigrolysis is sometimes very difficult to distinguish from normal variations, and has by itself given results as divergent as those obtained by Bernheimer and by Bach.

As the investigations of Szentagothai and of Bender and Weinstein are the only two mentioned in the literature which have used this precise technique of localized stimulation of the third-nerve nucleus, we thought it would be interesting to repeat these experiments. For technical reasons we chose the cat.

Further research will have to be done to show that our results, in broad lines in agreement with those of Szentagothai and of Bender and Weinstein, can be extended to man and other species.

Technique

Under barbiturate anesthesia, the head of the cat is held in the Horsley-Clarke apparatus illustrated in the paper of Talbot and Marshall. The posterior and lateral parts of the calvarium are chipped off to the junction with the tentorium cerebelli behind, and to the frontal bone rostrally. After incision of the meninges, the two occipital lobes are progressively drawn apart exposing the superior colliculi whose posterior aspects are covered by the anterior and medial end of the tentorium cerebelli. This bony nib is resected to give a clear approach to the superior colliculi. Bleeding is sometimes important when one approaches the brain stem; this can be greatly reduced by ligating the sinus longitudinalis superior and the vein of Galen. No animals died during the experiments.

When the anatomic landmarks of the superior colliculus are clearly visible, the stimulating apparatus is put in place. We have used a unipolar electrode formed by a sharpened steel needle. The needle is electrically insulated with varnish except at the extreme tip.*

The electrode is driven in vertically (that is to say in a plane perpendicular to the plane: external auditory canal-infra-orbital margin) at various distances from the midline and going from the posterior limit of the superior colliculus forward over about 4 mm. If the distance from the midline exceeds 1 mm. the needle going downward does not meet the nucleus. When the needle is presumed to be in the region of the third-nerve nucleus, which, as an average, is 6 to 7 mm. below the level of the sulcus between the superior colliculi, the electrical stimulus is applied at different depths and the eyes are observed.

As stimulus we have applied threshold (7 volts) and suprathreshold potentials of spike form at 60 per second, cathodally.

To locate exactly the points stimulated, we have used a technique consisting of passing through the electrode as anode a direct current of 10 to 20 microamperes during 10 seconds. The exposed tip of the electrode deposits in the nervous tissue some iron that can readily be shown later (fig. 2) as a spot about 0.2 mm. in diameter.

We find that in this type of experiment, recording very slight movements of the eye elicited by threshold stimulus, it is better to take out the orbital hooks of the Horsley-Clarke apparatus. These hooks strongly compress the inferior eyelid and conjunc-

* Tygon paint, U.S. Stoneware Co., Akron, Ohio.

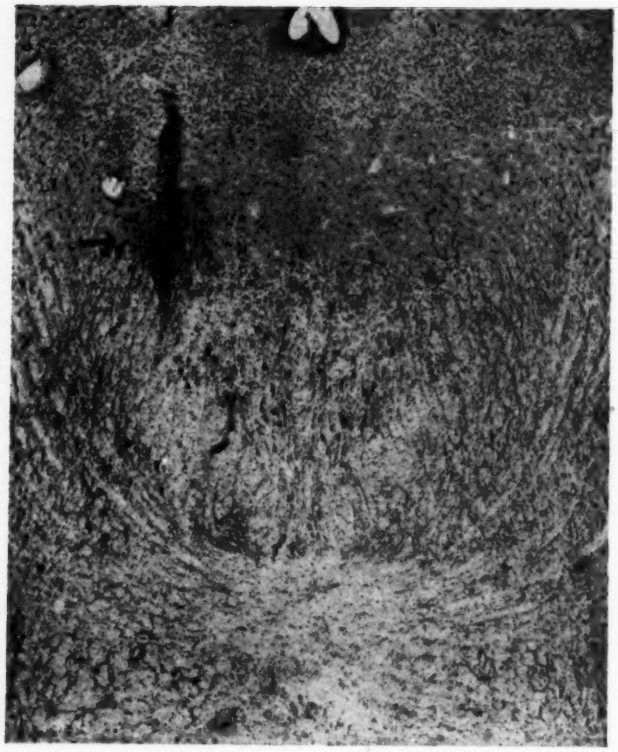

Fig. 2 (Danis). Frontal section at the anterior end of the third-nerve nucleus of the cat. (Gallocyanin stain, ×20.) The arrow indicates the iron blue mark. The dark line above this mark corresponds to the needle track. On the section it appears brownish.

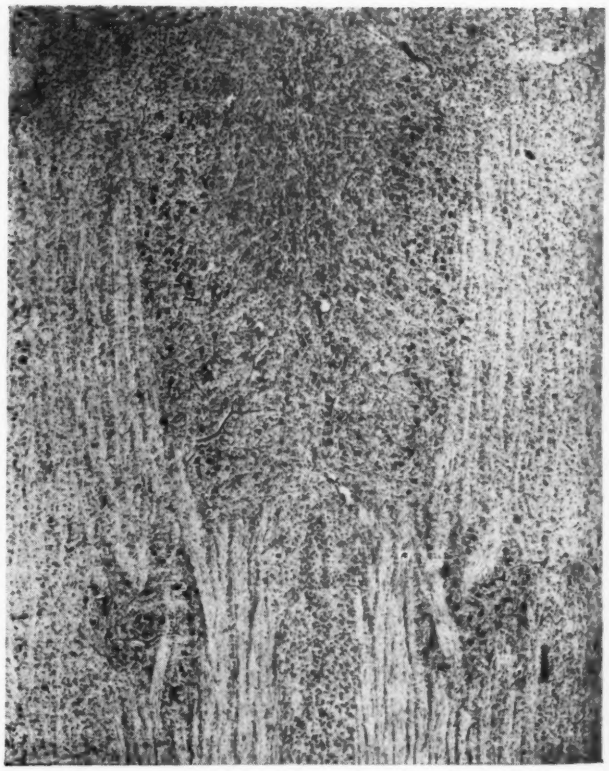

Fig. 3 (Danis). Horizontal section of the third-nerve nucleus of the cat. The section is parallel to the floor of the aqueduct of Sylvius. (Gallocyanin stain.) One can see the two lateral columns more or less fused on the midline. On the midline in the anterior part, the nucleus of Edinger-Westphal. Posteriorly and a little more laterally than the third-nerve nucleus, the two fourth-nerve nuclei. (× 24.)

tiva against the orbital rim and may, therefore, hinder completely free motion of the eyes. Before putting the stimulating apparatus in place, we replaced the orbital hooks by a frontal band which pressed the head down against the maxillary pieces to obtain a firm fixation.

At the end of the experiment the animal was killed and fixed by perfusion.

The brainstem is sectioned frontally in a plane approximately parallel to that followed by the electrode. Serial sections are made at 50 microns with the technique of Marshall, then submitted to a Prussian-blue test and stained with gallocyanine.

Anatomy

We will not give a detailed description of the oculomotor-nerve nucleus in the cat. This can be found in the papers of Abd-el-Malek Shafik, Bach, Beauvieux, Benjamin, Crouch, Latumeten, LeGros Clark, Levinsohn, Monakow, Panegrossi, Spitska, and Winkler.

The general structure is best seen on a horizontal section parallel to the floor of the aqueduct of Sylvius (fig. 3). The nucleus is formed by two columns of large multipolar cells, in anteroposterior direction under the floor of the aqueduct of Sylvius lying on the dorsal aspect of the posterior longitudinal bundle. In their anterior portion the two columns are clearly separated by a space containing the small-cell nucleus of Edinger-Westphal. In agreement with Bach, Benjamin, Brouwer, Latumeten, Levinsohn, Spitzka, and Winkler, and contrary to Beauvieux and Panegrossi, this nucleus was always seen to be present and well developed in the cat. In their posterior portion, the two masses get thicker laterally and dorsoventrally, and the central space is occupied by cells similar to those of the lateral columns; at this level there is no clear anatomic limit between the right and the left nucleus (LeGros Clark). At the posterior end, the two nuclei are once more clearly separated from each other. Like the other authors, we have not the impression that these medial cells are organized in a group anatomically distinct from the lateral nuclei, equivalent to the nucleus of Perlia.

Like Bach and MacLean, we have not found with enough constancy a subdivision of the lateral column in a dorsal and a ventral part, on different slides of the same or different animals, to justify a subdivision into two groups, ventral and dorsal, contrary to Abd-el-Malek Shafik, Monakow, Panegrossi, Spitzka, and Winkler.

On frontal sections the two columns have a V shape, opening upward. In the anterior part, the branches of the V are separated from each other; as one goes toward the posterior end the two branches tend to meet in their medial and inferior part as if by filling the space with cells similar to those of the lateral nucleus. These cells constitute the central group described on the horizontal sections.

Results

I. Points of stimulation

In a given animal, going on any transverse line from the right to the left, we have never seen any modification of the type of ocular response. If at a given level one obtained an elevation of the eye, at all other points of positive stimulation on the same transverse line and the same depth, the response was an elevation. The same applies to adduction of the eye or depression.

If one takes care to use a minimal stimulus, one will obtain a unilateral response—except when the needle is on the medial plane or so near that it is difficult to exclude a bilateral stimulation.

As far as the possibility of a contralateral innervation is concerned, our experiments do not allow us to exclude definitely this possibility, but it is certainly of little importance. In the large majority of cases the ocular response is ipsilateral, and the few cases where a contralateral response was obtained corresponded to stimulation on the midline. Because the needle is so near

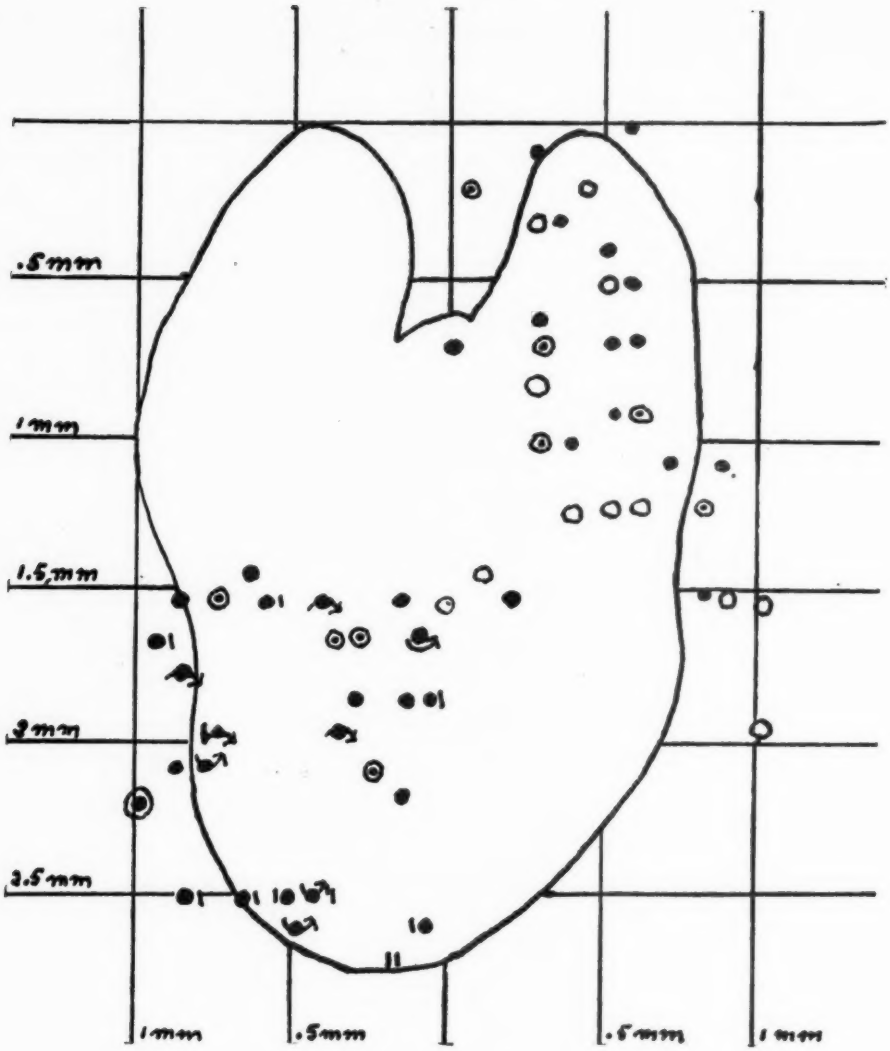

Fig. 4 (Danis). Horizontal projection at the depth of the nucleus (above the fibers level) of responses obtained in different cats (8):

● Represents an elevation of eye if on the left part of the drawing, a depression if on the right side (the point on the midline corresponds to a depression.

○ Represents a movement of pure adduction of the eye.

⊙ Represents a mixed movement of the eye, adduction with elevation if on the left part of the drawing, adduction with depression if on the right side.

Indicates an elevation of the upper eyelid.

Indicates if the elevation was associated with an intorsion.

Indicates if the elevation was associated with an extorsion.

This drawing gives the location of the points of stimulation in the antero posterior and the transverse plane.

the midline, the experimental conditions do not permit a discrimination between a contra- or bilateral innervation or a stimulation of the contralateral cells.

This agrees with the descriptions of Abd-el-Malek Shafik and Beauvieux. For these authors there are more direct than crossed fibers, the crossed fibers being located the nearest to the midline.

In Figure 4 the location of the different points of stimulation is given on a horizontal section. The fact that on the same anteroposterior level two different responses are sometimes seen is due to the

fact that it is a composite drawing of the points of stimulation recorded in eight cats and reduced to the same scale.

II. Types of response

As far as an organization in a dorsoventral direction is concerned, which was defended by Latumeten for the cat and Le Gros Clark for all the species as a general rule, we have been unable to find any definite proof.

We shall in connection with the work of Bender and Weinstein mention here a special point of technique. In their paper Bender and Weinstein describe the ocular responses obtained in the anterior part of the nucleus by stimulating points in dorsoventral direction: first depression of the eye, underneath adduction, still deeper elevation of the ipsilateral eye; between these three regions they obtained mixed responses: depression and adduction between the two first ones, elevation and adduction between the two deeper.

We have obtained the same type of responses in the anterior part of the cat's nucleus, but each time we have seen that the two inferior marks were in the efferent fibers of the third nerve while the point for depression of the eye was within the nucleus. The anteroposterior organization of the nucleus and the disposition of the radicular fibers must be kept in mind. The axones of the cells go downward, forward, and slightly outward to emerge at the inferoventral aspect of the peduncle. Thus the fibers pass under the cells anterior to those from which they are originated. This tends to be more marked on the Horsley-Clarke apparatus. The electrode being perpendicular to the orbitoaural plane forms with the longitudinal axis of the brainstem an angle of about 110° toward the rear. This explains furthermore that in front of the nucleus, the electrode can stimulate fibers without going through the nucleus. This can simulate a nuclear response if a careful location of the point of stimulation is not done (fig. 5).

This interpretation of some of the results of Bender and Weinstein, as applied to the cat, seems to be confirmed by the fact that in the anterior part of the nucleus, three different ocular movements could be obtained on the same dorsoventral axis and in always the same sequence—depression, adduction, elevation; in the middle part two responses only could be obtained—adduction and elevation; and in the posterior part only one—elevation.

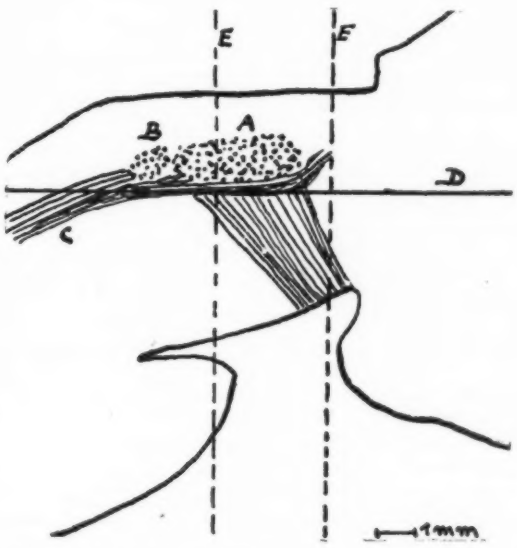

Fig. 5 (Danis). Sagittal section of the brainstem. The roof of the aqueduct of Sylvius is not represented. Composite drawing to show on the same plane the disposition of the third-nerve nucleus and the direction of the nerve fibers: (A) third-nerve nucleus; (B) fourth-nerve nucleus; (C) posterior longitudinal bundle; (D) horizontal plane of the Horsley-Clarke apparatus corresponding to the orbitoaural plane in the animal; (E) direction of the electrode.

In no instance, basing ourselves only on landmarking by the technique mentioned above, have we been able to obtain two points located on top of each other and still within the nucleus that would give different ocular responses. In some instances one could obtain at one point a sort of mixed response that could be attributed to the combined stimulation of two different muscles; but this always happened in zones that were at the common limit of two zones, disposed

anteroposteriorly, where one obtained a pure response of one or the other type.

III. Analysis of results

If in the interpretation and the mapping of the results, one remembers the features that we have mentioned, so as to retain only the points of stimulation located in the nucleus and not under it, a very definite organization of the third-nerve nucleus appears in the anteroposterior direction. Going from the rostral to the caudal end, one has successive points giving depression of the eye (isolated or sometimes with extorsion), then pure and extreme adduction of the eye, then elevation of the eye. The elevation of the eye when the stimulation is carried into the posterior part of the nucleus is accompanied by an elevation of the upper lid. An isolated elevation of the upper lid is only seen when the extreme posterior part of the nucleus is stimulated.

We have already mentioned that in certain cases a mixed response was obtained, depression of the eye with adduction or elevation with adduction. Plotting at the same scale the results obtained on different cats, one sees that these mixed responses fall at the margin of the zones where simple responses of one or the other type were obtained (figs. 4 and 6). It seems, therefore, that one can attribute these responses to a double stimulation due to the fact that the electrode is at the junction of two territories with different functions. Our technique shows no well-cut limit between two territories functionally different.

In their principal lines these results confirm those of Szentagothai and of Bender and Weinstein.

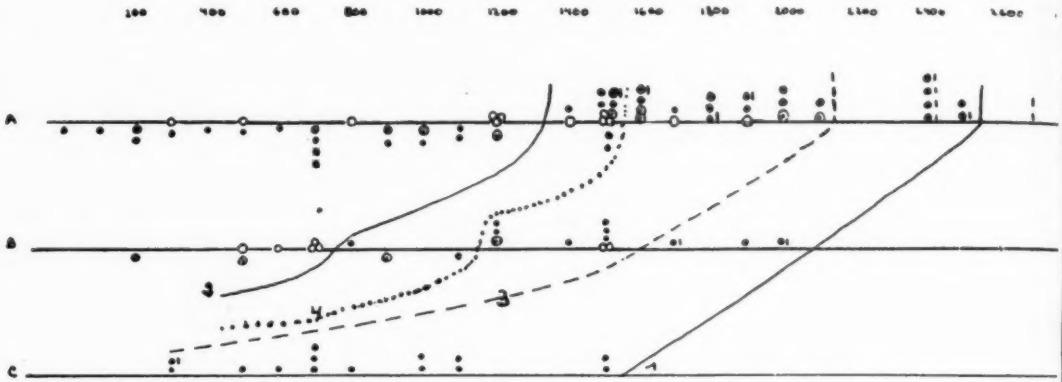

Fig. 6 (Danis). Anteroposterior projection of the point of stimulation observed in eight cats (only the points shown on Figure 4 are recorded) and reduced to the same scale. The numbers on the upper line indicate in microns from the anterior pole of the nucleus the level of the stimulation in the anteroposterior direction. The three horizontal lines correspond to three different levels in depth, the upper one corresponding to the intranuclear stimulations. In this projection the position of the stimulation in regard to the midline is not taken in account. All the points on the same horizontal line correspond to stimulation at the same depth in different animals. The number of points at a given level on a horizontal line corresponds to the number of different points of stimulation. All the points on the same vertical line correspond to the same situation of the electrode, and the horizontal line indicates their respective level in depth and the response obtained. For every point on (C) and (B) there is point on (A).

● Indicates an elevation of the eye or a depression, if it is above or under the horizontal line respectively.

○ Indicates a pure movement of adduction.

⊙ Indicates a mixed movement of adduction with elevation if above the horizontal line, or depression if under the horizontal line.

\ Indicates an elevation of the upper lid.

1 and 2 indicate the posterior and anterior limit of the zone where an elevation of the eye is obtained; 3 indicates the posterior limit of the zone where an adduction could be obtained; 4 indicates the posterior limit of the zone where a depression could be obtained.

We have, however, obtained rather different results as far as the localization of the inferior oblique is concerned. Bender and Weinstein localized with some doubt the radicular cells of this muscle in the anterior part of the nucleus between the ones for the inferior rectus and the ones for the internal rectus. They base this opinion on the fact that, at this point, stimulation gives an isolated extorsion of the ipsilateral eye (in 4 monkeys out of 24 experimented on). Szentagothai gives the same localization for the territory in front of the one for the internal rectus, but this was nearly always accompanied by a slight depression of the eye. In no cats have we obtained elevation of the eye by stimulation of points between the territories for the internal rectus and the inferior rectus. In all cats the same sequence of movements was obtained, going from the caudal to the rostral end of the nucleus—elevation, adduction, depression.

We believe that the inferior oblique must be localized behind the internal rectus in the

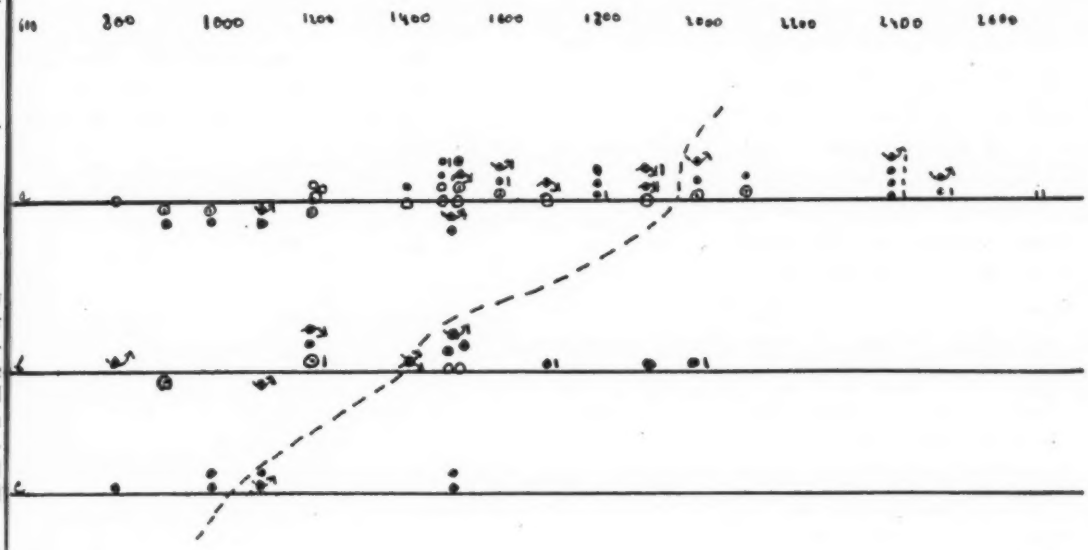

Fig. 7 (Danis). Same projection as Figure 6 to show the distribution of the movement of elevation with intorsion and of elevation with extorsion.
Elevation with intorsion (superior rectus).
Elevation with extorsion (inferior oblique).

inferior oblique (fig. 1) in the cat, basing his opinion on the same criteria, isolated extorsion of the eye.

This argument does not seem decisive because at this level we are at the posterior limit of the zone for the inferior rectus, a muscle which also gives an element of extorsion in the depression of the eye (this was also found by Bender and Weinstein). It seems, therefore, doubtful to use the extorsion of the eye as only criterion to differentiate the action of the inferior oblique from the inferior rectus.

We too have obtained extorsion in the vicinity of the superior rectus and levator palpebrae because, in this zone, we could obtain three types of elevation—elevation with intorsion (superior rectus), pure elevation (it was impossible to see any torsional component in this movement), and elevation with extorsion (inferior oblique). We may analyze a representation (fig. 7) which superimposes the results obtained at similar anteroposterior levels in three different cats. Of the seven points for the inferior oblique (elevation with extorsion) five are well posterior to the average locus for the superior rectus (elevation with intorsion). These

few data show the center for the inferior oblique is even behind that for the superior rectus.

Conclusions

In general our results on the cat are in agreement with those of Szentagothai and of Bender and Weinstein done respectively on the cat and dog and on the monkey. However instead of localizing the inferior oblique in the anterior part of the nucleus between the inferior rectus and the internal rectus, we locate it behind the internal rectus in the vicinity of the superior rectus and probably between the superior rectus and the levator palpebrae, near its antagonist the superior oblique.

1. With the technique used there is no functional organization of the third-nerve nucleus in the transverse nor in the dorsoventral direction. The latter we regard as a confusion of nuclear and fiber organization.

2. The majority of cells send fibers to the ipsilateral muscles. A contralateral innervation was not seen, but the hypothesis of a weak crossed innervation is impossible to deny by this method.

3. The functional organization of the third-nerve nucleus follows the following plan, going from the caudal to the rostral end: inferior rectus, internal rectus, superior rectus, inferior oblique, levator palpebrae. This does not correspond to a discrete anatomic organization, for the experiments with stimulation show that these zones merge into each other.

These experiments give a functional organization of the third-nerve nucleus in the cat that differs from those proposed on the basis of retrograde degeneration, by Abd-el-Malek Shafik and Latumeten, for the cat, and by Brouwer for man. This scheme in its broad lines resembles the one demonstrated by Szentagothai for the cat and by Bender and Weinstein for the monkey.

Of the other experimenters using the technique of retrograde degeneration, Monakow, Bach, and van Biervliet proposed a roughly similar arrangement of the third-nerve nucleus with the depressor muscles in the rostral part and the elevator muscles in the posterior part. All three authors also put the inferior oblique behind the internal rectus in the vicinity of the superior rectus, but only van Biervliet localized, in the rabbit as we do in the cat, the inferior oblique behind the superior rectus.

The author wishes to acknowledge his indebtedness to Dr. Samuel A. Talbot, whose help, interest, and criticisms have permitted the realization of this work.

References

Abd-el-Malek Shafik: On the localization of nerve centres of the extrinsic ocular muscles in the oculomotor nucleus. J. Anat., **72**:518-523, 1938.

Bach, L.: Zur Lehre von den Augenmuskellähmungen in den Störungen der Pupillbewegung. Arch. f. Ophth., **47**:339-386; 551-630, 1899.

Beauvieux: Les origines réelles du moteur oculaire commun chez le chat. Arch. d'opht., **46**:401-410, 1929.

Bender, M. B. and Weinstein, E. A.: Functional representation in the oculomotor and trochlear nuclei. Arch. Neurol. & Psychiat., **48**:98-106, 1943.

Benjamin, J. W.: The nucleus of the oculomotor nerve with special reference to innervation of pupil and fibers from the pretectal region. J. Nerv. & Ment. Dis., **89**:294-310, 1939.

Bernheimer S.: Experimentelle Studien zur Kenntnis der Innervation der inneren und ausseren vom Oculomotorius versorgten Muskeln des Auges. Arch. f. Ophth., **44**:481-525, 1897.

van Biervliet: Noyau d'origine du nerf oculomoteur common du lapin. La Cellule., **16**:1, 1898.

Brouwer, B.: Klinisch-anatomische Untersuchung über der Oculomotoriuskern, Ztschr. f. d. ges. Neurol. u. Psych., **40**:152-193, 1918.

Crouch, R. L.: Efferent fibers of Edinger-Wesphal nucleus. J. Comp. Neurol., **64**:365-373, 1936.

Latumeten: Ueber de kernen van den nervus Oculomotorius. Thesis, Utrecht, 1924.

LeGros Clark, W. E.: The mammalian oculomotor nucleus. J. Anat., **60**:426-448, 1926.

Levinsohn, G.: Beitrag zur Physiologie des Pupillenreflexes II. Arch. f. Ophth., **59**:436-458, 1904.

MacLean, A. J.: Attempt to identify central cells mediating kinesthetic sense in extrinsic eye muscles. Arch. Neurol. & Psych., **17**:285-302, 513, 1927.

Marshall, W. H.: An application of the frozen sectioning technique for cutting serial sections through the brain. Stain Technol., **15**:133-138, 1940.

von Monakow, C.: Gehirnpathologie. Vienna, A. Hölder, Ed. 2, 1905, p. 103.

Panegrossi, G.: Weiterer Beitrag zum Studium der Augenmuskelnerven-Kerne. Monatschr. f. Psychiat. u. Neurol., **16**:268, 1904.

Schwabe, H.: Ueber die Gliederung des Oculomotoriushauptkerns und die Lage der den einzelnen Muskeln entsprechenden Gebiete in demselben. Neurol. Centralbl., no. 17, 1896.

Spitzka, E. C.: The oculomotor centers and their coordinators. J. Nerv. & Ment. Dis., **15**:413, 1888.

Szentagothai, J.: Die innere Gliederung des Oculomotoriuskernes. Arch. f. Psychiat., **115**:127-135, 1942.

Talbot, S. A. and Marshall, W. H.: Physiological studies on neural mechanisms of visual localization and discrimination. Am. J. Ophth., **24**:1255-1263, 1941.

Winkler, C.: Opera Omnia. t. VIII. Ed. Bohn. 1927.

ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS*

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Essential progressive atrophy of the iris is a relatively uncommon disease. Duke-Elder[1] characterizes it as a unilateral disease of unknown etiology that is marked by a slowly progressive atrophic change in the tissues of the iris, which leads to the complete disappearance of large portions of the iris and ultimately ends in blindness from absolute glaucoma.

Since Sugar[2] reported his case in 1945, Arruga[3] and Cyukrasz[4] have each added a new one, the latter author reporting a bilateral case in which cyclodialysis successfully relieved the glaucoma. This paper brings the total cases reported in the literature to 51. Undoubtedly more cases have existed and it is hoped that some of these will be reported. The writer has had the opportunity to follow a case of this disease for over 10 years and the case history is being submitted as an addition to the current literature.

The iris atrophy and the glaucoma are both the outstanding clinical observations in this entity.

CASE REPORT

History. Miss N. S., aged 22 years, was first seen by me on June 5, 1934, with the complaint that the pupil in her right eye had "dropped down." There was no history of trauma or disease. Her vision had not been affected. Examination disclosed a pear-shaped pupil, which was eccentrically displaced down, giving the impression of a congenital coloboma of the iris. Uncorrected vision was 20/50 in the right eye, and 20/200 in the left eye. She had a myopic error and correction improved vision in the right eye to 20/30; the left eye to 20/20.

Examination on June 14th and 19th of the same year revealed no change; the media were clear and the fundi were normal but there were deep physiologic cups in the discs. The patient was advised to return at frequent intervals. In October, 1934, she had a bilateral salpingo-oophorectomy, the pathologic records of which could not be obtained.

Course: She failed to return for observation until March 14, 1939, five years later. On that day she stated that one week previously she noticed that there were two pupils in her right eye. Examination showed the displaced pupil below and a large gap in the superior iris. The gap had many strands of iris tissue in it. The nasal portion of the iris was gray, while the normal iris tissue was dark brown. The true pupil reacted to light, especially in its upper portion.

Vision was: O.D., 20/100, correctible to 20/40 with a −1.50D. sph.; O.S., 20/100,

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* From the Long Island College of Medicine and the Brooklyn Eye and Ear Hospital. Presented before the Brooklyn Ophthalmological Society, October 23, 1947.

correctible to 20/20−1 with a −1.25D. sph. The peripheral fields were normal at this time. Tension was; O.D., 23 mm. Hg; O.S., 15 mm. Hg (new Schiøtz). Wassermann test was negative; nose and throat examinations were essentially negative; but a positive Mantoux reaction was obtained.

On April 18th, the iris atrophy had extended farther nasally and the gap had increased in size (fig. 1). The lower margin of the true pupil was adherent and immobile. The patient was warned to expect

Fig. 1 (Lasky). *April 18, 1939.* The iris atrophy had extended farther nasally and the gap had increased in size.

trouble and to report at the first sign of pain or inflammation in the eye.

We next saw her on September 7, 1939, (fig. 2). By then she had developed more visual loss and had some redness of the eye with a vague feeling of disturbance. The tension was found to be: O.D., 35 mm. Hg; O.S., 20 mm. (new Schiøtz), and the peripheral field of the right eye showed a moderate general contraction with an angular defect in the inferior nasal quadrant. The blind-spots were normal. Pilocarpine solution (1 percent) was prescribed for use every four hours in the right eye.

Surgery. On September 16th, she returned complaining of a heavy dull pain in the right eye. Tension proved to be: O.D., 56 mm. Hg; O.S. 18 mm. Operation was advised and the patient consented. In view of the deterioration of vision and poor prognosis in this ailment, it was decided to do a Lagrange sclerectomy with as large an opening as possible. The procedure was accomplished without any difficulties on September 21st, and a large dark bleb resulted. Tension became: O.D., 23 mm. Hg; O.S. remained at 18 mm. No changes were noted in the iris or pupil of the right eye. The eye gradually became white, and a good bleb, partially filled with black pigment, was present. The fundus was clearly seen but the view was distorted due to a large astigmatic error, which appeared following the operation.

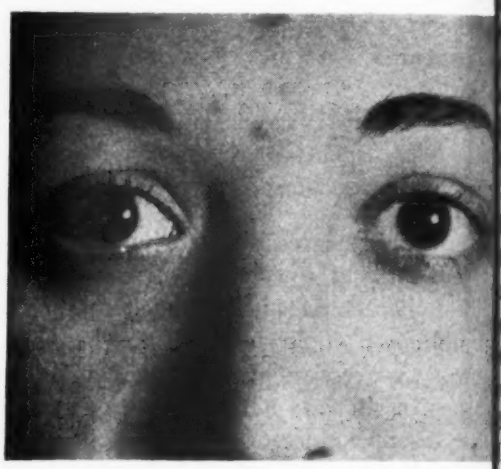

Fig. 2 (Lasky). *September 7, 1939.* By now the patient had developed more visual loss and had some redness of the eye with a vague feeling of disturbance.

There was occasional pain in the right eye despite normal tension. The bleb became progressively darker and more prominent. Iris atrophy continued to progress and there was a grossly evident anterior synechia formation at the 6- to 7-o'clock region of the limbus. On February 17, 1940, almost five months after the Lagrange sclerectomy, the tension of the right eye rose to 48 mm. Hg (new Schiøtz), so an Elliot trephining operation (2 mm.) was performed temporally to the previous opening. However, this proved adequate for only six months A second Elliot trephination (2 mm.) was done at the 3-o'clock limbus position. After this procedure, the tension remained at 35 mm. Hg (new Schiøtz).

Because of the failure of these various operations, it was decided to abandon the anterior-segment procedures and to do a

posterior sclerectomy. On November 8, 1940, a 2-mm. button of sclera was removed from the infero-temporal quadrant of the globe in the region of the equator. This procedure was accompanied by the loss of a small amount of vitreous. A large bulbous area appeared beneath the conjunctival flap. It remained boggy for about two months and gradually became reduced in size. The tension did not return to normal limits until the operative area had become well healed with a flat well-formed bleb. On January 18, 1941, tension was: O.D., 13 mm. Hg (Schiøtz).

Outcome. The patient was not seen again by me until November 5, 1941. At this time the vision was: O.D., 1/200; O.S., 20/50; correctible to O.D., 20/30 with a −1.0D. sph. ⌒ −8.0D. cyl. ax. 70°; O.S., 20/20 with a −1.0D. sph. Tension was: O.D., 25 mm. Hg (Schiøtz). The original pupil was displaced to the 7-o'clock limbal area. The Lagrange filtration was marked by a large irregular black bleb. The two Elliot trephinations could hardly be discerned. The site of the posterior sclerectomy was flat but thickened. The lens and vitreous were clear allowing a view of a normal fundus, although it was a greatly distorted picture due to the large astigmatic error.

The situation was relatively unchanged on March 8, 1942. Vision in the right eye was still 20/30 with correction, and tension was 20 to 25 mm. Hg (Schiøtz).

On April 15, 1947, the patient presented herself again for an examination and the following findings were elicited. Vision was: O.D., 15/400, unimprovable; O.S., 20/30, improved to 20/20 by −0.75D. sph. Tension was: O.D., 27 mm. Hg; O.S., 15 mm. Hg (Schiøtz). (She stated that tension in the right eye had been 25 to 27 mm. Hg (Schiøtz) at all times since 1942.) The left eye was normal in all respects. The right globe (fig. 3) showed very little evidence of the posterior sclerectomy, just some conjunctival thickening. At the site of the Lagrange sclerectomy, there was a large irregular bleb that was tense and well filled with black-stained material. The Elliot trephination on the nasal side was flat and sealed. The iris was adherent anteriorly from the 6- to 7-o'clock position at the limbus; the entire upper portion was missing except for a few long strands that tapered from the iris below to the anterior lens surface above; the

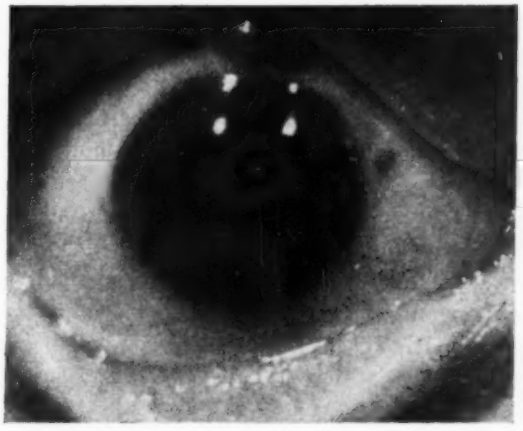

Fig. 3 (Lasky). *April 15, 1947.* The iris was adherent anteriorly from the 6- to 7-o'clock position at the limbus; the entire upper portion was missing except for a few long strands that tapered from the iris below to the anterior lens surface above.

remnant of iris had a serrated edge and hung like a hammock from the 8- to 3-o'clock position. The pupil was somewhat triangular with the base above; this base still reacted to light. There were no deposits on the posterior surface of the cornea. The aqueous was clear.

The lens was studded on its anterior and posterior surfaces by numerous pigment particles ("bird-in-flight" type) that were especially dense near the nasal equator. A posterior cortical lens opacity was present below. Beneath the site of the nasal Elliot trephination a notch in the lens edge was clearly visible. The vitreous seemed clear and the fundus appeared to be normal except for a pale disc.

Comment

The general consensus of opinion at present is that the glaucoma is secondary to the

formation of peripheral anterior synechias, Lloyd.[5] However, various other theories have been advanced; such as Feingold's[6] conception of irritating substances formed as a result of iris tissue destruction; Kreiker's[7] idea that iris tissue debris occluded the chamber angle; Licsko's[8] coupling of the latter with loss of iris surface for resorption of aqueous. Rochat and Mulder[9] first advanced the thought that the anterior synechias are the agents responsible for the glaucoma. Rones[10] points out that many instances occur in which large segments of iris are lost by operation or trauma and glaucoma does not necessarily supervene.

Iris atrophy has been a moot question. De Schweinitz[11] believed it to be due to a local abiotrophy. Feingold suggested that a congenital vascular disturbance of the smaller iris circle caused the atrophy; Sugar explained that a lack of involvement of the sphincter militates against this view. Kreiker believes that in adult life the cytolitic process that occurs in embryonic life, causing the resorption of the pupillary membrane, suddenly becomes newly active and attacks the normal iris tissue. However, I am inclined to concur with Rochat and Mulder in their belief that the iris tissue atrophy is secondary to the constant, progressive stretching of the iris as it is pulled to one side of the anterior chamber. As has been described in the present case, there has been formed a mass of anterior synechias not only in the chamber angle below but also recently above to the visible cornea; there is a constant inexorable traction force. The longer the eye survives, the more anterior synechias form and the greater is the iris tissue destruction.

The true cases of "essential" atrophy of the iris have all been notable for the absence of any posterior synechias, iris inflammation, or any signs of an inflammatory process other than the anterior synechias that are a constant feature. The answer to the problem appears to lie in the presently unknown factor which initiates the first localized anterior synechia. In this case the extensive migration of iris pigment to the posterior surface of the lens, as well as to the anterior surface, indicates the marked degree of mobilization of this substance in the "essential atrophy."

One Nevins Street (17).

References

1. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, C. V. Mosby Co., 1941, v. 3, p. 2404.
2. Sugar, H. S.: Glaucoma and atrophy of the iris. Am. J. Ophth., **28**:744-748, 1945.
3. Arruga, H. A.: Case of essential atrophy of the iris. Arch. Soc. oftal. hispano-am., **5**:233-237 (April) 1945.
4. Cyukrasz, I.: A case of bilateral genuine iris-atrophy, Brit. J. Ophth., **31**:176-179 (Mar.) 1947.
5. Lloyd, R. I.: Heterochromia and allied conditions, Am. J. Ophth., **14**:287-299 (Apr.) 1931.
6. Feingold, M.: Essential primary atrophy of iris. Am. J. Ophth., **1**:1, 1918.
7. Kreiker, A.: Zur genuinen atrophie der iris. Klin. Monatsbl. f. Augenh., **80**:492, 1928.
8. Licsko, A.: Irisatrophie nach glaucom. Klin. Monatsbl. f. Augenh., **71**:456, 1923.
9. Rochat, G. F., and Mulder, W.: On progressive atrophy of the iris with formation of holes and glaucoma. Brit. J. Ophth., **8**:362, 1924.
10. Rones, B.: Essential atrophy of the iris. Am. J. Ophth., **23**:163-171 (Feb.) 1940.
11. De Schweinitz, G. E.: Essential progressive atrophy of the iris. Arch. Ophth., **56**:10, 1927.

SENILE MACULAR LESIONS*

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ATROPHY OF THE CHOROID

The choriocapillaris and the pigment layer of the retina, with the lamina of Bruch between them, are referred to frequently as the tapetum. This is a convenience sanctioned by use and the fact that to the ophthalmoscopist they are a single tissue. The tapetum returns the light from the ophthalmoscope to the examiner's eye and hides the deep choroidal vessels.

In older persons the tapetum becomes depigmented and, as a rule, exposes the sclerosed choroidal vessels which finally become opaque. This atrophic process begins in the nasal periphery and may or may not involve the macula in the late stages.

In addition to exposure of the choroidal vessels, a sheen or watered-silk effect spreads over the macula, interfering somewhat in the examination of that part of the fundus. This sheen is due to changes in the internal limiting membrane. It is surprising how far atrophy of the tapetum may go with but moderate effect upon the vision other than contracted fields and enlarged blindspots. Perhaps this may be explained by the work of Cohen and Verhoeff disclosing normal caliber of the retinal and choroidal vessels despite advanced changes in their vessel walls. A more advanced stage of this atrophy is featured by a denser sheen over the macula, a meaty fundus background with undulating finer retinal vessels, indicating a wrinkling of the tissue beneath. The vision now is definitely lower and retinal changes can be seen with red-free light (fig. 1).

Fig. 1 (Lloyd). Early stage of choroidal atrophy in a woman, aged 61 years. There is a hole in the macula after a slight injury. Vision is 20/40.

*Read in part at the 82nd annual meeting of the American Ophthalmological Society, San Francisco, California, June, 1946.

The atrophic process may spread over the entire fundus exposing the deep sclerosed vessels everywhere except at the macula and, as long as this patch of choroid remains, vision is very good indeed in *good light*. If the patch beneath the macula disappears, the patient is blind. Some of these

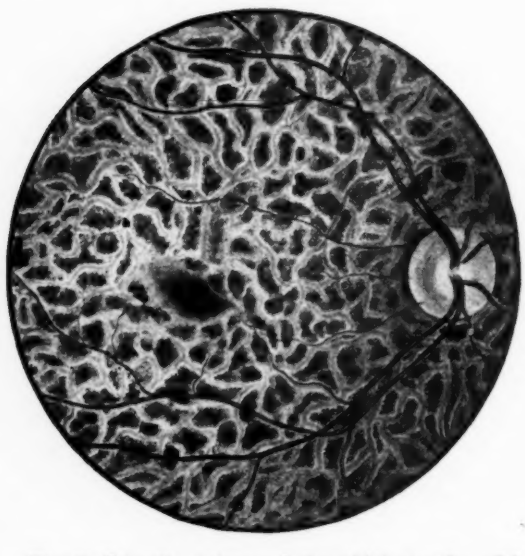


Fig. 2 (Lloyd). Advanced choroidal atrophy with surprisingly good vision in strong light (20/25 and 20/33). The patient, a woman, aged 64 years, showed night blindness and ring scotomas.

patients do not complain of night blindness, but some cannot see in poor light, and some also have ring scotomas (fig. 2). This unusual fundus picture may be referred to as progressive choroidal atrophy (fig. 3); or it may be designated by the term, choroideremia. None of these eyes have come to the laboratory as far as I can learn, and the cases are so few that essential details are lacking. No family incidence has been shown in this group, distinguishing these cases from those in which one or more members of a family have pigmentary retinitis or night blindness with fundus pigment and secondary posterior capsular cataract.

General choroidal atrophy may be contrasted with two more localized forms, peripapillary and central. The peripapillary atrophy is most frequently seen in high myopias which run through families. The central type is referred to as central areolar atrophy and may possibly develop without adequate explanation but is more often seen as the late stage of pseudotumor and angioid stripes. It follows occlusion of vessels in the nerve or retina after injections about the face, basal fractures, and without discovered etiology.

Hole in the macula

This is rarely seen except with choroidal atrophy and is at first limited to the tapetum with later serious effects upon vision. A rarer sequella of choroidal degeneration is pseudotumor of the macula caused by an accumulation of serum or blood beneath the pigment layer followed by characteristic changes.

Pseudotumors of the macula

These lesions, which require differentiation from tumor, are modest elevations of

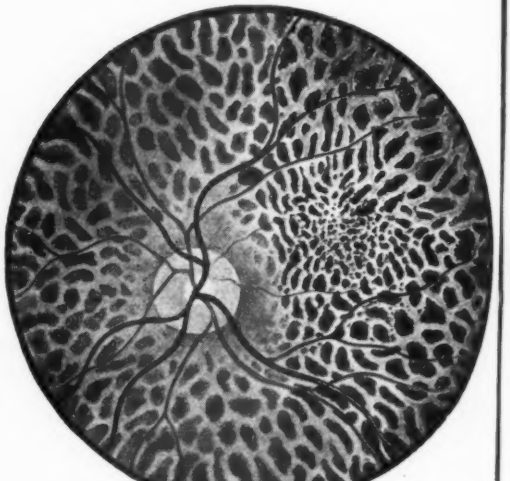


Fig. 3 (Lloyd). Late stage of progressive atrophy of the choroid. This man, aged 65 years, was blin but gave no history of difficulty in seeing at night

3 to 6 diopters and of 6 to 8 disc diameter in width and height, somewhat lighter i color than the background and easily mis taken for a patch of choroiditis unless th

binocular scope is used. The first complaint, loss of vision, may precede objective evidence by a few weeks. The first visible change may be small hemorrhages, followed soon by the rounded mass in the macula (fig. 4). Two patients have recovered from this stage but neither had normal vision and one had a relapse within a year. The lesion may remain in the original state for some time but most of them undergo further changes within a few months. Cyst formation (fig. 5) is the most common type of lesion and is probably due to the clear exudate breaking through the pigment layer of the retina leaving no other covering than the retina. Less common is the organization of the exudate to form the woolly masses of Coats's disease (figs. 6 and 7). Better known is the sheet of organized tissue beneath the retina, the disciform degeneration of the macula (Kuhnt-Junius). Cysts are unstable and do not usually remain for any length of time (fig. 8). The clear exudate within may disappear along with the tapetum, leaving a bare spot in the macular area with sclerosed vessels and atrophic choroidal tissue as the background. Occasionally the sclera may be exposed. The fluid may spread laterally and raise the retina in folds, producing a flat detachment (fig. 9) of limited proportions. After a time, the involved tapetum atrophies and an extensive area of thinned pigmented tapetum remains. Occasionally there are streaks of "retinitis striata" beneath the retinal vessels (choroiditis proliferans, Fuchs).

The hand scope does not give satisfactory details of the cyst because the retinal image is never sharp but obscures the view of the deeper parts. The binocular scope shows the diaphanous retina in advance of the pigmented and hemorrhagic tapetum beneath. None of these spreading defects have been seen to pass over into a typical retinal detachment of average extent or elevation but I believe it does occur in elderly persons.

Judging from the appearance of the macular region after reattachment of the retina and the microscopic changes in the pigment layer in the late stages of pseudotumors, one should not expect too much improvement of vision because of damage to the pigment epithelium and the choriocapillaris.

Choroidal hemorrhages and the masses of "rice grains" (fig. 9a) about the macula usually referred to as retinitis circinata are common to every stage of these lesions. To the hand scope, the "star figure" of hypertensive disease, wreaths of retinitis circinata, and many hemorrhages seem to be in the retina but are actually in the tapetum.

A fulminating type of pseudotumor is due to the choroidal hemorrhage breaking through the lamina of Bruch, then through the pigment layer of the retina so it is seen with nothing but the retina over it. These lesions smother the light of the ophthalmoscope and look like the dark masses seen after cataract extraction which are recorded as subchoroidal hemorrhage or detachment of the choroid. The appearance of these is so alarming that the eye is usually enucleated at once.

One such case, reported by Dr. Verhoeff and Dr. Grossman, flattened down after six months; the macular region lost color; and microscopic examination showed the organized subretinal tissue characteristic of disciform degeneration. A case of the kind came to my care and, although the diagnosis was in doubt, because retroillumination at operation showed complete opacity of the area involved, the eye was removed about one week after total loss of central vision, known to have been 20/20 before the attack (fig. 10).

In none of the slides examined from cases of this type or illustrated in the literature, was the choroid stripped from the deeper tissues nor was blood found beneath the choriocapillaris. Excepting the stripping of the choroid by rupture of long ciliary vessels during operation for cataract or glaucoma, the term subchoroidal hemorrhage or detachment of the choroid is a misnomer.

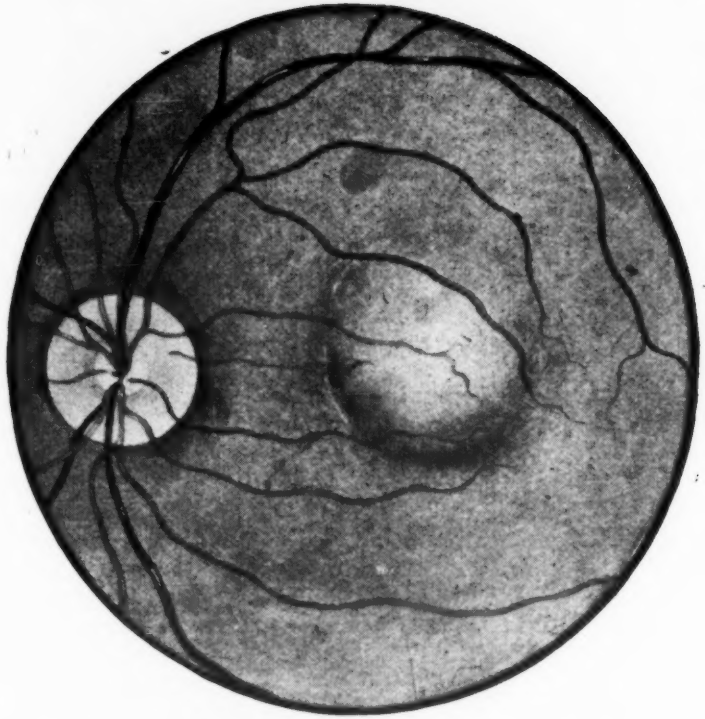


Fig. 4 (Lloyd). This illustrates the first stage in the development of pseudotumors of the macula. The patient, a man, aged 41 years, had recently been treated for syphilis. Small hemorrhages, followed soon by the rounded mass in the macula, were the first visible changes.

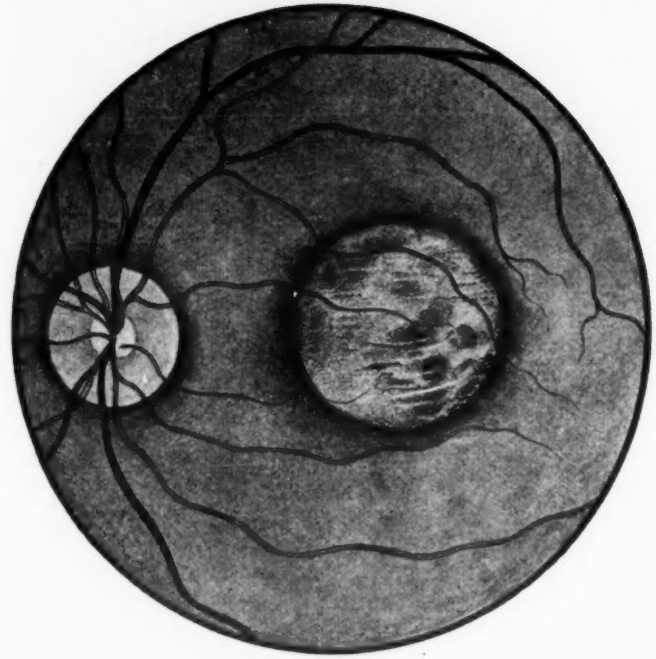


Fig. 5 (Lloyd). The same eye as shown in Figure 4. This drawing, made six months later than Figure 4, illustrates the change from a typical lesion to the cystic stage.

Fig. 6 (Lloyd). The patient, aged 54 years, showed as the first lesion, a hole in the macula, followed by hemorrhage and pseudotumor one year later.

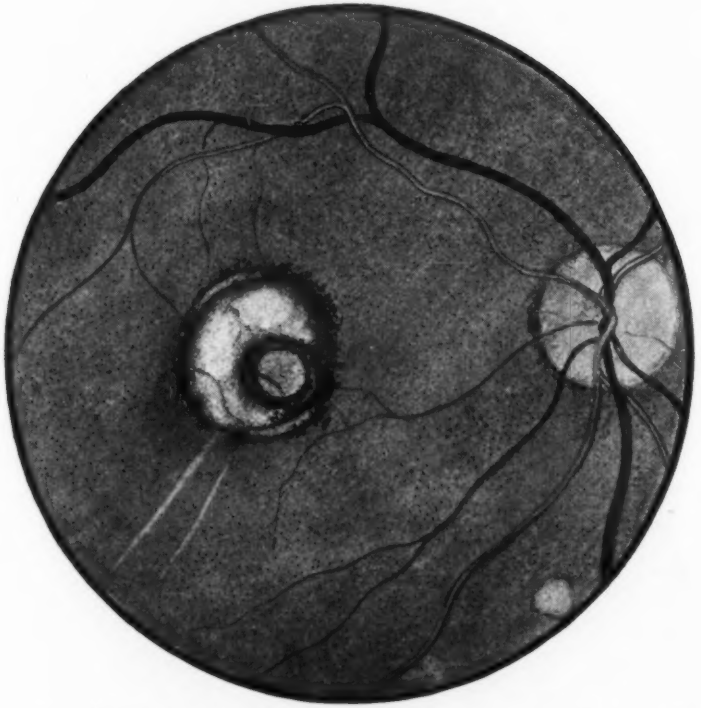
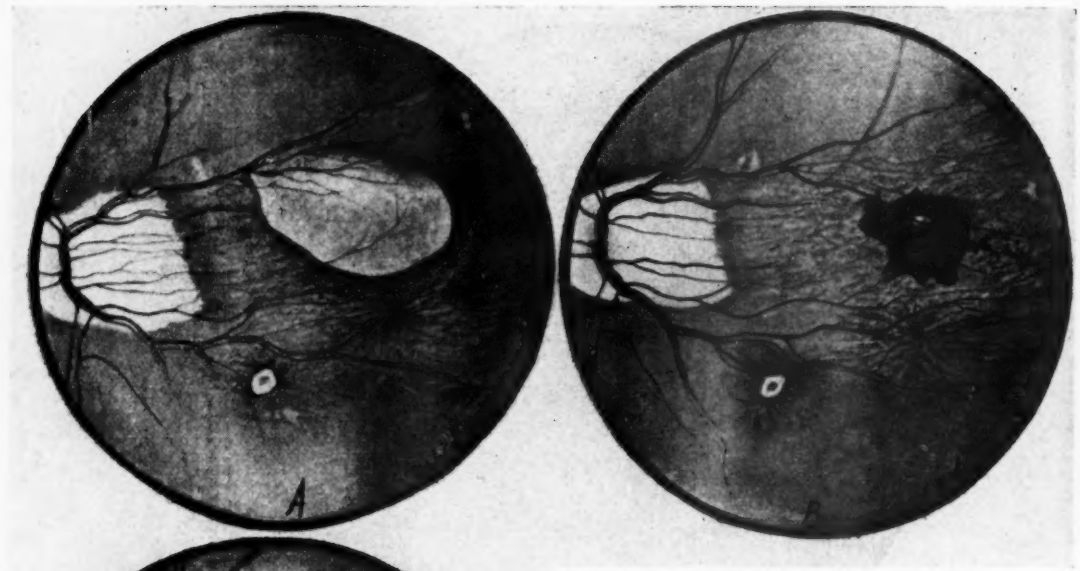


Fig. 7 (Lloyd). This drawing of the same eye as is shown in Figure 6 was made two years later. It illustrates the change into Coats's disease.



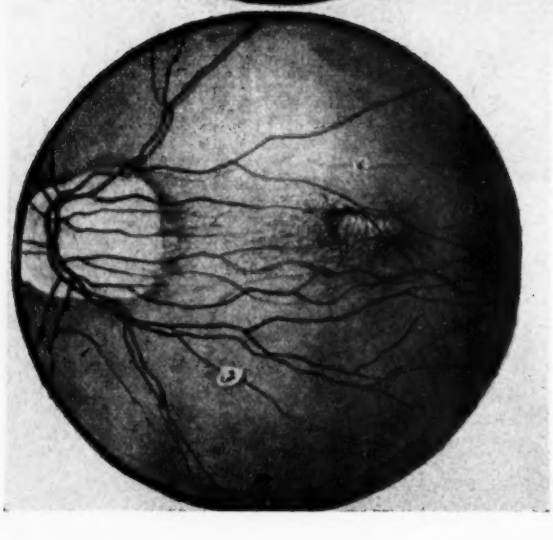


Fig. 8 (Lloyd). This illustrates the change from a cystic lesion (A) to a very small atrophic area in seven months. The patient, past fifty years of age, was highly myopic when the lesion appeared. (B) The lesion (−16D. sph. ⊃ −1.50D. cyl. ax. 165° = 20/50, 2 letters). Vision dropped to 1/200 three months later. (C) Five months later. Vision is 20/100. (Dr. Paul Chandler.)

The difference between the hemorrhagic type of pseudotumor, the usual pseudotumor, and the cyst which often follows is the presence of blood beneath the retina in the first instance; clear fluid beneath the pigment layer in the second; and clear fluid beneath the retina in the third. Judging from the appearance of the spreading lesion after cyst formation, it is fair to say that the difference in appearance between a cyst and a retinal detachment is due to the character of the retina at the macula and elsewhere.

The pseudotumor occurs most often in

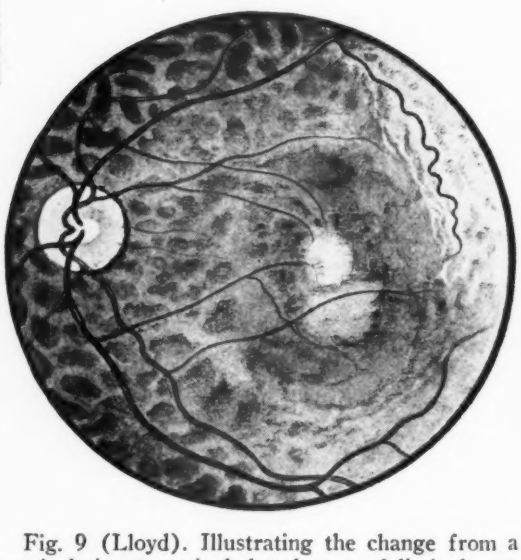


Fig. 9 (Lloyd). Illustrating the change from a cystic lesion to retinal detachment of limited area in six months.

older patients of the "healthy" arteriosclerotic type quite independent of the retinal lesions of the cardio-vascular-renal syndrome, although the two may go together. The lesions are often bilateral, with an interval of time elapsing between the attacks. The patient with pseudotumor may live for years after losing central vision, in contrast to those affected with high blood pressure. Quite young patients have been reported as having this condition, but the youngest in this group was aged 34 years. Most cases are found in the 50- to 60-years age group.

There is no explanation for the vulnerability of the macula that the location and frequency of the lesion implies. Atrophy of the peripheral tapetum with sclerosis of the choroidal vessels is a common antecedent and in three cases there were also holes in the macula. One patient had a definite history of syphilis.

Behr asserts that the lamina of Bruch controls the passage of nourishment and the return of waste products from the choriocapillaris to the external retinal layers. A highly albuminous fluid accumulates beneath the pigment layer, stripping it from the lamina. The pigment cells react to the exudate much as lens stroma reacts when exposed to aqueous. The epithelial layer proliferates and degenerates, making a sort of capsule beneath the retina with especially thick margins which throw back the light from the scope to form the image perceived by the examiner as retinitis circinata (Seefelder).

Fig. 9a (Lloyd). Bilateral lesions (see also Figure 9b) in a man, aged 65 years. "Retinitis circinata," here illustrated, is often seen in these cases.

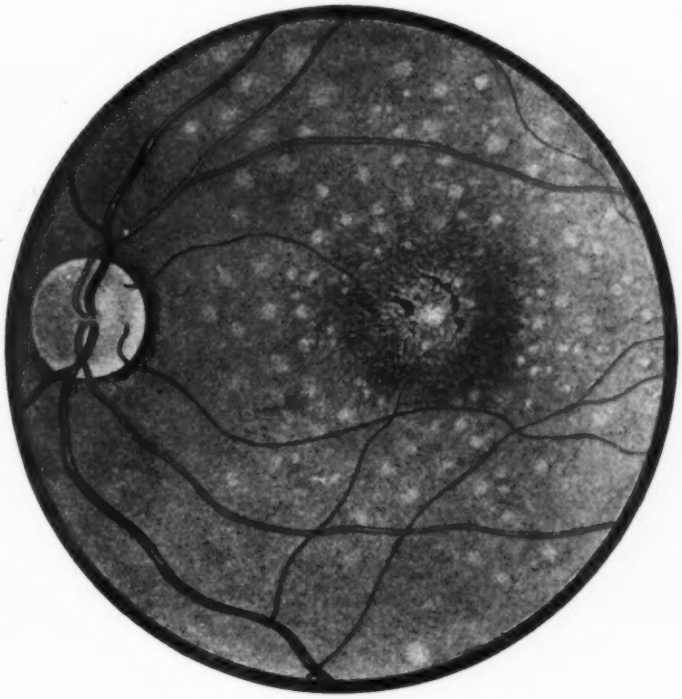


Fig. 9b (Lloyd). The soft spots in this eye are colloid masses in the lamina of Bruch.

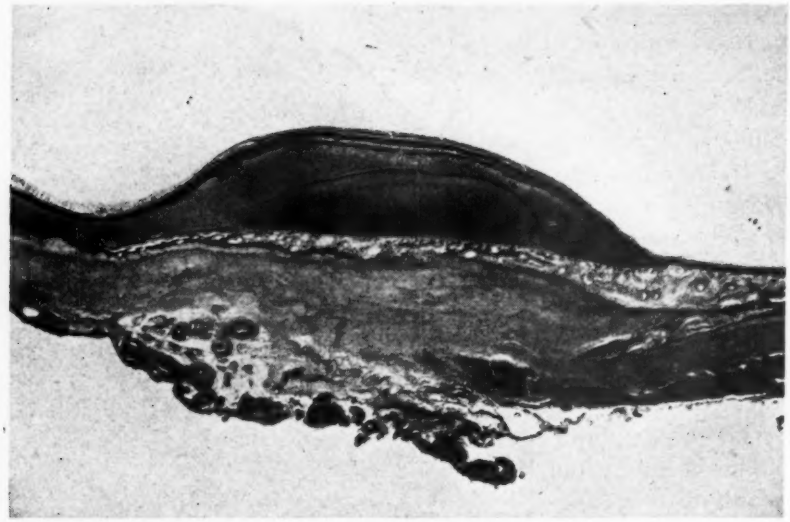


Fig. 10 (Lloyd). Sudden loss of vision with a dark mass in the macular area looking like "detachment of the choroid" after cataract extraction. The eye was removed one week after onset. The hemorrhage came from the choriocapillaris, lifted and broke the pigmentary layer. The break through the pigment layer is at the left.

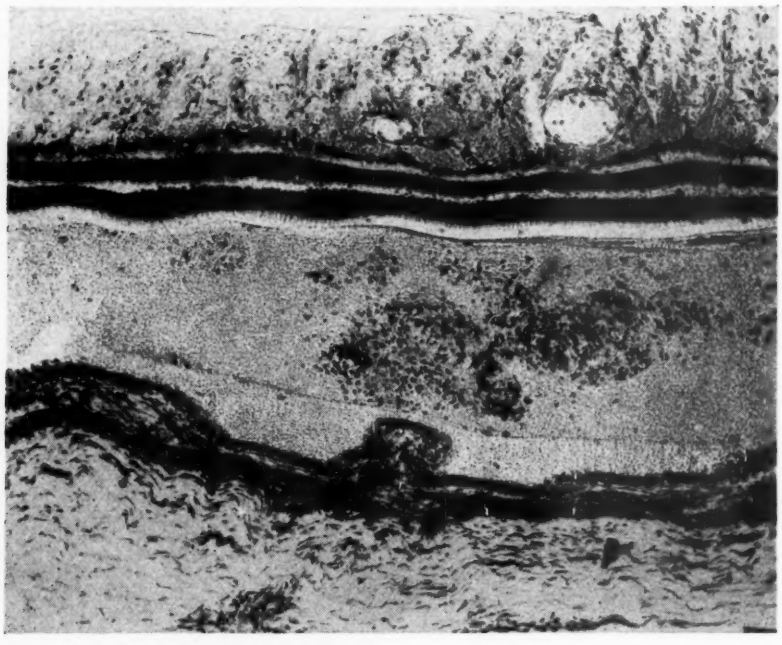


Fig. 11 (Lloyd). "Bud" from choriocapillaris passing through the lamina into the subretinal collection of blood in an acute case. From the same eye as shown in Figures 6 and 7.

Behr explains the connective tissue, bone, and cartilage found in the exudate beneath the retina in Coats's disease and disciform degeneration of the macula (fig. 11) as a metaplasia; but Axenfeld, Verhoeff, Vogt, Sandoz, and E. V. L. Brown* have demonstrated breaks in the lamina of Bruch (fig. 12) through which vessels passed into the organized subretinal tissue. This vascular intrusion also explains the recurring choroidal hemorrhages so often seen in these cases. Once vessels have entered the subretinal space, any form of connective tissue may develop. The diagnosis is confused by metastatic tumors of the choroid which look just like innocent lesions. This is probably due to the fact that the tumor mass in the choroid is small and concealed by an accumulation of subretinal fluid.

Every case should be examined carefully for sources of metastasis (fig. 13), the most frequent being the female breast and the prostate gland. Metastatic tumors at the macula are very rare and, in these older individuals, there is time enough to study the case until the diagnosis is certain. In younger patients, the ophthalmoscopic picture may be so strange and suggestive of tumor that we must fall back upon the well-established rule that an eye without possibility of useful vision should be sacrificed if there is serious doubt of the nature of the lesion.

Chorioretinal tuberculosis presents a very similar picture but the vitreous is involved early and the course of the disease is more rapid. Scars of previous attacks and involvement of retinal vessels may assist in establishing the diagnosis. Positive reaction to tuberculin tests is also of diagnostic value.

Fig. 12 (Lloyd). The black lines indicate the lamina of Bruch through which an arteriole is passing to the organized subretinal tissue. (From, Verhoeff and Grossman: Pathogenesis of disciform degeneration of the macula. Tr. Am. Ophth. Soc., 1937, p. 262.)

*I am indebted for the privilege of examining slides from the three cases reported by Drs. Verhoeff and Grossman and also one from the case reported by Axenfeld in 1915. Drs. E. V. L. Brown, Beulah Cushman and Arnold Knapp have kindly given me slides from cases of this type reported by them.

Syphilis has rarely entered the picture because the fundus lesions it produces are results of vessel degeneration rather than gumma.

Fig. 13 (Lloyd). Metastasis from carcinoma of the breast in a patient about fifty years of age. The lesion was improved by X-ray therapy, but the other eye was involved in two years. (Dr. Walter Hipp.)

When these symptoms appear in children (fig. 14), the question of birth injury will arise. Lesions resulting from injury at birth are stationary, with threads of new tissue extending into the vitreous from the organized mass following hemorrhage. Blunt trauma may produce most confusing pictures but the damage is so severe that the eye is rarely retained. If it is retained, however, examination of the fundus is usually precluded by damage to the cornea or organization of tissue in the vitreous.

Two cases of congenital retinal folds (falciform ablation) required some study, but this condition is always connected with the disc and, although the fundus shows marked deviation from the normal, the clear vitreous permits satisfactory fundus examination.

A somewhat similar lesion is the so-called preretinal edema. A patient with this type of macular lesion had lived for many years in Africa and complained of poor vision in one eye after returning to this country. He improved under treatment, but I did not have the opportunity to follow the case to conclusion. The binocular scope located the pigment dots and the bubbles in the edematous retina but an exudate beneath the pigment layer caused the elevation. With the hand scope, the lesion looked as if the collection of fluid was preretinal, like the so-called preretinal hemorrhage, but both are really beneath the retina. Neither of these conditions has been examined under the microscope and, until this is done, there will be differences of opinion. I am sure

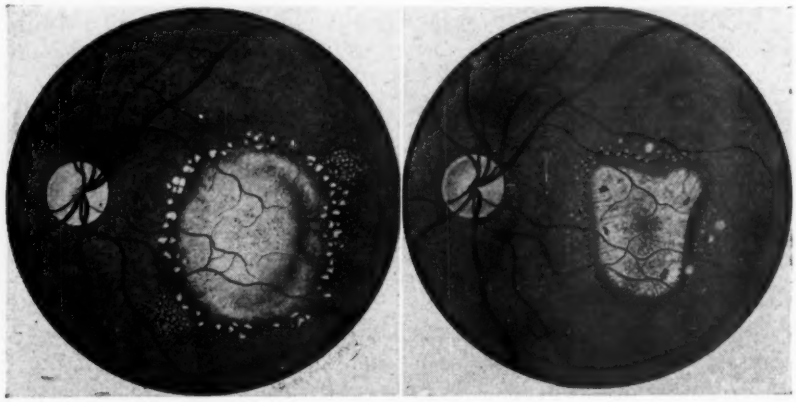


Fig. 14 (Lloyd). Lesion in a child aged 7½ years. The interval between the two drawings is two years. Tests were negative. The large spots about the lesion in the illustration at the left are crystals.

the so-called preretinal edema is not preretinal and would suggest use of the binocular scope in such cases to clear the doubt.

Angioid striping

This is a degenerative process occurring in eyes of older persons but with notable exceptions to age limits. The familial tendency is well known and, although this is considered an entity, the pathologic process is very similar to that of pseudotumors. It is not unusual to find typical cases in a family, as well as cases with similar fundus details excepting the stripes. The breaks in the lamina of Bruch are numerous but, because of brittleness, many are made when the section is cut.

The literature contains many case reports, but very few pathologic examinations have been made because only eyes removed after death have come to the laboratory. Hagedoorn, Böck, and Bertha Klien have each presented completed reports of cases studied with the ophthalmoscope and serially sectioned in the laboratory. Accumulation of fluid beneath the pigment layer, with organized subretinal tissue like that found in disciform degeneration, has been found but no mention is made of vascular intrusions.

An illustration (fig. 15) is presented showing angioid stripes in one eye while the other has a large atrophic area in the macular region with exposed sclerosed vessels of the deep choroid.

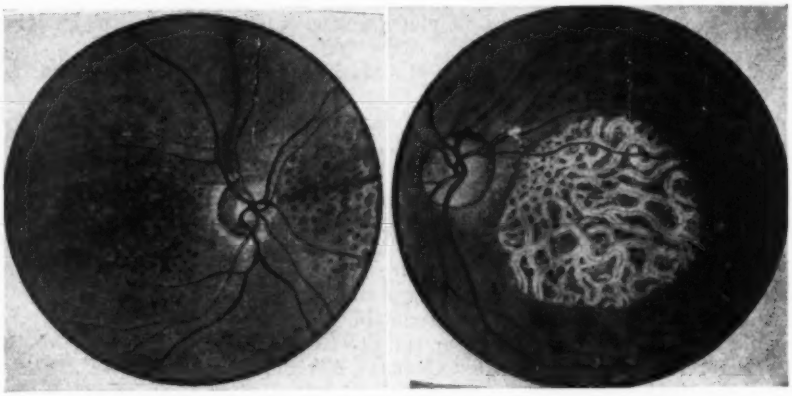


Fig. 15 (Lloyd). Angioid stripes in the right eye and central areolar choroidal atrophy in the left.

Summary

From this review it seems that a number of characteristic fundus lesions, considered to be entities, are really stages of a degenerative process affecting mainly the lamina of Bruch, the vessels of the choriocapillaris, and the pigment layer. This is to be expected, since Time takes its toll first of the elastic tissues, and its effects upon the vessel walls, skin, the sphincters and the detrusors are well known. The lamina is an elastic tissue and the choroidal vessels in the macular area seem especially vulnerable.

Although no detachment of the area or size seen in typical cases has occurred under observation, the similarity to flat detachments seen in young persons with myopia of moderate degree suggests the importance of routine serial sectioning of all posterior fundus lesions, with a systematic search for breaks in the lamina of Bruch and vascular intrusions.

Because of the reaction of the pigment layer to accumulations of fluid beneath it, one suspects that diathermy is effective because it inhibits the secretion and not because it closes a tear. Also, involvement of the macula in a detachment would seem to occlude much hope of good vision after reattachment.

These opinions should not lead one to think that I am opposed to operation for detachment. An ophthalmologist who has practiced when detachments were treated by prolonged rest in bed, subconjunctival salt injections, compress bandages, and tapping of the subretinal space—with a recovery rate of zero—can appreciate the benefits of the modern operation. The binocular microscope is essential to a satisfactory study of cases of this type and anyone who has used it can readily appreciate the advantages of observation with two eyes.

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References

Adler: Disciform degeneration of the macula. Arch. Ophth., **30**:379, 1933.

———, and Scarlett: Juvenile macular exudative choroiditis. Arch. Ophth., **31**:144, 1944.

Axenfeld: Retinitis exsudativa externa mit knochenbildung in sehfahigem Auge. Arch. f. Ophth., **90**:452, 1915.

Batten: Peculiar symmetrical swellings in macular region. Tr. Ophth. Soc. U. Kingdom, **34**:217, 1904.

Behr: Zur Anatomie der scheibenformigen Makuladegeneration. Klin. Monatsbl. f. Augenh., **83**: 109, 1927.

———: Beitrag zur Anatomie and zur Pathogenese der scheibenformigen Degeneration am hinteren Augenpol. Ztschr. f. Augenh., **75**:216, 1931.

Böck: Zur Klinik und Anatomie der gefässahnlichen Streifen im Augenhintergrunde. Ztschr. f. Augen., **95**:1, 1938.

Brown: Retroretinal tissue from choroid in Kuhnt-Junius degeneration of macula. Arch. Ophth., **23**:1157, 1940.

Coppez and Denis: Retinite exsudative maculaire senile. Arch. d'Ophth., **40**:129, 1933.

Cushman: Disciform degeneration of the macula. Am. J. Ophth., **11**:720, 1928.

Elschnig: Pseudotumor of the macula. Klin. Monatsbl. f. Augenh., **62**:145, 1919.

Feingold: Senile external exudative macular retinitis. Tr. Am. Ophth. Soc., **22**:268, 1924.

Gissy: Ein weiterer Fall von "preretinalen Oedem." Ztschr. f. Augenh., **57**:423. 1925.

Guist: Ueber "preretinalen Oedem." Ztschr. f. Augenh., **54**:37, 1925; **55**:269, 1926.

Hagedoorn, A.: Angioid streaks. Arch. Ophth., **21**:746, 1939.

Hanssen: Anatomy of disciform degeneration of the macula. Ztschr. f. Augenh., **62**:360, 1930.

Horniker: Ueber eine Form von zentraler Retinitis auf angioneurotischer Grundlage. Arch. f. Ophth., **123**:286, 1929.

Holm: Retinitis exsudativa externa. Klin. Monatsbl. f. Augenh., **59**:319, 1930.

Junius and Kuhnt: Die scheibenformige Entartung der Natzhautmitte. Berlin, S. Karger, 1926.

Kahler and O'Brien: Disciform degeneration of the macula. Arch. Ophth., **13**:937, 1935.

Kitahara: Clinical observations of chorioretinitis centralis serosa, frequently seen in Japan. Klin. Monatsbl. f. Augenh., **97**:345, 1936.

Klien: Angioid streaks. Am. J. Ophth., **30**:955, 1947.

Knapp: Subretinal exudate simulating sarcoma of the choroid. Arch. Ophth., **48**:359, 1919.

Kraupa: Die Retinitis centralia annularis. Ztschr. f. Augenh., **1**:335, 1923.

Lawford: Subretinal new growth of doubtful nature. Tr. Ophth. Soc. U. Kingdom, **31**:257, 1911.

Oguchi: Eine besondere Form von Chorio-Retinitiscentralis, mit nachbildartiges Sketom. Arch. f. Ophth., **110**:25, 1922.

Paul: Choroiditis exudativa unter dem Bilde der scheibenformige Entartung der Netzhautmitte. Ztschr. f. Augenh., **63**:205, 1927.

Pillat: Ueber hintere Glaskorper abhebung. Ztschr. f. Augenh., **57**:335, 1925.

Sandos: Histology of Bilateral Pseudotumor of Macula. Vogt, Festschrift, 1939, p. 97.

Sattler: Sarkomahnlicher Aderhauttumor herbeigefuhrt durch ein retrochoroidealen Bluterguss bei einem an Retinitis circinata errinern den Krankheitsbild. Klin. Monatsbl. f. Augenh., **90**:199, 1933.

Seefelder: Arch. f. Ophth., **120**:230, 1928.

Verhoeff and Grossman: Pathogenesis of disciform degeneration of the macula. Tr. Am. Ophth. Soc., **35**:262, 1937.

Vogt: Klin. Monatsbl. f. Augenh., **95**:93, 1935.

von Graefe: Ueber centrale recidivierenda Retinitis. Arch. f. Ophth., **12**:211, 1866.

Walsh and Sloan: Idiopathic flat detachment of the retina. Am. J. Ophth., **19**:195, 1936.

Weintraub: Ein Fall eines umschriebenen, verganglich, Oedema der Makulagegend. Klin. Monatsbl. f. Augenh., **82**:808, 1929.

Wolfflin: Pathologische Anatomie der Retinitis exsudativa externa. Arch. f. Ophth., **117**:33, 1926.

BARRIERS AND AIDS TO FUSION*

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In attempting to discuss fusion, one is led from the study of the simple reflex to the elaboration of final percepts and concepts—from physiology to the realm of metaphysics. As objective investigators, we may not like the subjective aspects of psychology, but we should at least remember that the study of science must be synthetic as well as analytic. Thomas Young, the physicist and ophthalmologist, himself taught and followed that truism. Hence we shall not avoid mentioning such metaphysical concepts as consciousness, attention, and association, even though we translate them to forms of cerebral functioning.

SOME CONCEPTS OF FUSION ATTAINMENT

In considering the various theories of fusion in the visual cortex, the most easily understood are those which propound certain anatomic fusion centers, or a physiologic centering of fibers from corresponding retinal areas.

Unlike his well-known theory of conditioned reflexes, Pavlov's proposed thesis of cortical excitation and irradiation has not proved acceptable.

The replacement theory is summarized by stating that the cortical center selects parts from maximum capacity uniocular images, so that the final image is a mosaic derived from replacement rather than addition.

Chavasse suggests two possible theories. The first stipulates that fusion occurs by selection from two uniocular cerebral patterns, and the second that it occurs by reciprocal cerebral inhibition so that only one pair of corresponding areas can function at the same time. His refusal to consider the sensory aspects of fusion, however, and his insistence on regarding it simply as a motor reaction brought about by reflex activity leaves his reflexes like an army without a commander with no explanation of integration or synthesis.

The conclusions of Sherrington have led to the most complete concept of fusion. He proved that each uniocular image is developed and elaborated independently and that the union of the two images is the product of synthesis derived from interpretation with other already elaborated sensations.

We know that fusion is of late phylogenetic development and, as such, is easily liable to modification or perversion. For its natural and normal functioning it is necessary to have an overlap of the visual fields of the two eyes, a highly organized central nervous system, corresponding retinocerebral elements, and a correctly functioning extraocular muscle system so that the visual axes are correctly oriented.

The neural image arising in the retina is transmitted through nerve impulses to the lower visual reflex centers and the appropriate motor reaction for fusion is initiated by the higher visual reflex centers. We may note here the general acceptance of the conclusion that the semidecussation of the fibers at the chiasma has a motor significance rather than a sensory significance, since it provides a common efferent path. Thus there is no necessity for a common representation in the cortical field (Sherrington, Duke-Elder, and others).

BARRIERS TO FUSION

Barriers to fusion may be divided into two classes—those which prevent the development of fusion and those which cause its breakdown. Specifically they may be listed as follows:

* Presented at the sixth annual meeting of the American Association of Orthoptic Technicians, Chicago, October 12, 1947.

1. Dioptric. (a) Due to unequal uniocular images—for example, anisometropia over 4D, high astigmatism, aniseikonia. (b) Refractive—for example, hypermetropia or myopia may predispose to convergent or divergent strabismus respectively, the cause being a too tightly conditioned accommodation-convergence reflex. The uncorrected hypermetrope overaccommodates to see clearly and consequently overconverges. The resulting diplopia causes him to converge more to separate the images. Conversely the myope has no need to accommodate and, therefore, underconverges producing divergent strabismus. The initial treatment lies in correction with glasses, followed by an orthoptic loosening of the too tightly associated accommodation-convergence link.

2. Anomalous retinal correspondence. Although this is certainly a barrier to fusion, it should be considered as a biologic adaptation rather than a cause per se of the nondevelopment of fusion. The theory that anomalous retinal correspondence is a conditioned reflex—as Chavasse presumes—would be a most satisfactory explanation, if one is prepared to accept the principle of sensory conditioning. However, in spite of alleged synesthesia, no sensory conditioning has yet been demonstrated. The classical conditioned reflex refers to a motor or secretory response.

Duke-Elder regards anomalous retinal correspondence as secondary acquired correspondence which is a functional attribute. As an orthoptist, I can testify that it can be cured in 4 to 24 treatments in over 50 percent of cases (under 12 years of age) preoperatively. Another 15 to 25 percent will obtain true retinal correspondence in the first weeks after operation, provided preoperative, bimacular stimulation has been given. The cure may involve the extinction of a conditioned reflex, the reinforcement of an inhibited neural pathway, or the grounding of new neural connections.

If the anomalous retinal correspondence is not corrected before operation, any of the following situations may ensue:

a. The patient may develop a new anomalous correspondence adapted to the new position of the eyes.

b. He may turn the eye in again to the original angle in order to restore the old anomalous correspondence.

c. He may have persistent diplopia of the paradoxical or false type, or of the true type.

d. The possibility of obtaining fusion without training is remote and might occur only if there was a history of previous fusion, and if the eyes were straight after operation. Even then suppression would probably prevent the development of fusion.

3. Amblyopia. In this condition we need only consider amblyopia ex anopsia, which may be either the cause or the result of strabismus. When the child is conscious of diplopia, he turns one eye in further to separate the images and then suppresses the vision in that eye so that it becomes amblyopic from disuse. This is an active inhibition which is cerebral in character. Treatment consists in appropriate stimulation of the amblyopic eye and is usually successful. Unless fusion is stimulated and attained before operation while the amblyopia is in the process of being cured, there will be no binocular hold after the operation and the diplopia-strabismus-suppression cycle will be repeated. It is conceivable that when the vision of an amblyopic eye has been improved by occlusion, without fusion training being given simultaneously, diplopia will become troublesome, and the child will consequently either develop anomalous retinal correspondence or increase the angle of squint.

4. Suppression. When total or partial dominance is given to the image of one eye, and there is a loss of physiologic retinal rivalry, the usual cause is either incompatibility of the uniocular images or heterophoria.

In the latter, a small central suppression may prevent symptoms at first, but when compensation or adjustment can no longer be maintained, the effort becomes conscious and symptoms appear. The cure in this case consists of antisuppression treatment and the development of increased fusional range to overcome the heterophoria.

In the case of an alternating strabismus, suppression is both intensive and extensive, and requires treatments to obtain simultaneous cortical response to each uniocular impression. Sherrington proved the existence of the neural states of excitation and inhibition, which are believed to be due to electrical changes in the cortex. Inhibition may be of the intrinsic or adaptative type, as opposed to the extrinsic or antagonistic type, and in cases of amblyopia and suppression is probably of both types.

5. Neurogenic anomalies such as nystagmus, spasm, and so forth.

6. Hyperphoria and hypertropia. A vertical deviation of over five diopters is usually beyond the range of fusion, and can thus be considered a barrier to fusion.

7. Loss of central fixation and false fixation. These are further perversions of the amblyopic group.

8. Deviation of the visual axes. Strabismus, whether it is convergent or divergent, is a barrier to fusion since a correctly functioning extraocular musculature is a primary requisite. Whatever the etiology of the strabismus, the visual axes must be restored to approximate parallelism by operation. Nevertheless, it should be emphasized that the previous anomalies, such as amblyopia, suppression, anomalous retinal correspondence, and so forth, must be corrected and a fusion ability initiated before operative procedure, or the patient will be presented with straight eyes and a true or paradoxical diplopia, or a suppression which will cause him to squint again. The end organs should be capable of receiving the images, and then the two uniocular images must be compatible and capable of independent elaboration in the visual cortex. The facilitation of appropriate pathways and the integration of higher centers will then effect a correct motor response when the external musculature is finally adjusted.

A squint is a conditioned reflex and for its extinction requires not only the correction of muscular changes by operation, but also reconditioning by training.

Before final discharge, the patient should have good fusion range, and be able to converge correctly.

To reiterate, we have given the various theories and a definition of fusion, discussed the barriers to fusion, and the possibility of their removal by refractive correction, orthoptic treatment, and operation. To illustrate the importance of orthoptic training as being of therapeutic value in addition to its diagnostic uses, it will now be necessary to investigate the visual anlage of the individual, and the learning process.

Neuro-anatomy of vision

In the young child we have a condition of flux and an histologically proved anatomic network indicating a richly branching and interlacing central nervous system—fertile ground for the future formation of new associations and habits or reflexes.

Unlearned automatic reflexes are innately present, and they may be either physical reflexes which are not registered in consciousness—for example, the pupillary light reflex—or they may be psycho-reflex in nature and are registered in consciousness—for example, convergence. There are also learned or conditioned reflexes, of which an example is the accommodation-convergence linkage.

The inherited cellular adaptation may be modified by experience, and indeed neural tissue possesses the property of modification if only in a small degree. Of primary importance, however, is the possibility of the facilitation of present pathways and the

grounding or establishment of new connections between neurons—between receptors and effectors.

A response may be evoked or reinforced by changes and modification at the synapses, thus achieving a decrease in the threshold. Since the nerve impulse along a fiber is believed to be "all or none," the changes in the threshold must occur at the synapse; that is, at the point of excitation, by bioelectric potential and resulting ionization, or by certain physiochemical changes in the nerve cell.

It is conceived by recent experimentation that the mechanism of the transmission of nerve impulses across the synapse does not differ qualitatively from that of their conduction along the fibers themselves. It is only quantitatively greater at the synapse since the neuronal surface itself is greater here, and the chemical and metabolic changes associated with electric changes, which occur at the neuronal surface, show a greater manifestation here during nerve activity. Other neural theories which seek to explain conditioning are briefly that learned action occurs in closed neural chains, as opposed to direct neural chains, or that it depends on bioelectric fields for organization.

The quality of summation needs to be mentioned here, since it reflects on orthoptic training, which seeks to evoke responses by stimuli and association. Temporal summation occurs when two successive stimuli to the same nerve evoke a response even if the stimuli are too weak or the threshold too high for them to act individually. Spatial summation occurs when two stimuli are applied to two different afferent nerves which play upon the same reflex center. Summation is greater when they are applied simultaneously, and at least two simultaneous impulses are necessary to excite a neuron.

The learning process

We accept the conditioned reflex of Pavlov, as a true basis for the learning process, but since it has been proved that a conditioned response may be effected even when the cortex is destroyed, and considering that a simple chaining of conditioned reflexes will not adequately account for the patterning and integration of habits in their complex presentation, we may understand the neurologists' refusal to accept conditioning as a total explanation of neural behavior.

For a more complete understanding of the problem we must consider the functioning of the higher centers through the factors of interest and attention, and their superimposition upon the reflex foundation by means of association paths. Sherrington has shown the primary importance of the higher centers as modifiers and controllers of the reflexes.

Skilled reactions are controlled reactions, and any training is, therefore, a modification of the activity of the higher reflex centers through conscious control. The association systems which are concerned with the complex sensorimotor integration of adaptative behavior are capable of modification by training. The process of encephalization—or the dominance of the cortex which occurs in phylogenetic development—is indicative of learning possibilities, even ontogenetically.

Role of orthoptics in fusion

The role of orthoptics in the development of fusion is thus accomplished by the following physiologic and neurologic considerations:

1. The elicitation of response by the facilitation of neural pathways through the use of correct stimuli; the facilitation signifying a nonlearned increase in the strength of the response.

2. The elicitation of response by means of conditioned reflexes. New reflexes or responses may be conditioned by means of the fixation reflex (surely a primary reflex par excellence) and by kinesthetic re-

flexes. The direct and simultaneous stimulation of the two maculas, and hand and eye coördination are fundamental aids.

3. The consummation of the reflex arc is in itself a direct intrinsic stimulation to further response, especially if it is in the nature of a biologically superior response.

4. The elaboration of two uniocular images in the cortex and the arousing of the factors of attention and interest so that, with increased permeability of the association pathways, fusion is elaborated in a final synthesis.

Summary

We have considered the theories of fusion and the necessary neural and physiologic factors. Reference has been made to the barriers to fusion and their removal by refractive, orthoptic, and operative means. The validity of orthoptic training on the basis of our present knowledge of the learning process is demonstrated and the attainment of fusion considered in the light of cortical neurophysiology.

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References

1. Chavasse, B.: Worth's Squint. London, Baillière, Tindall & Co., 1939.
2. Duke-Elder, W. S.: Textbook of Ophthalmology. London, Henry Kimpton, 1932, v. 1, pp. 1034-1035.
3. Fulton, J. F.: Physiology of the Nervous System. New York, Oxford Medical Publications, Oxford University Press, 1943, pp. 57-59, 331, 493, 501.
4. Hilgard, E. R., and Marquis, D. G.: Conditioning and Learning. New York, Appleton-Century Co., 1940, pp. 310-336.
5. Lancaster, W. B.: Ocular motility. Am. J. Ophth., **24**:485, 619, 741, 1941.
6. Pavlov, I.: Lectures on Conditioned Reflexes. New York, International Universities Press, 1928.
7. Sherrington, C. S.: Integrative Action of the Nervous System. New Haven, Yale University Press, 1906, p. 20.

Discussion of Miss Eyles' paper

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The neuron component of the ocular-muscular apparatus has been somewhat neglected in our work because it is so poorly understood even by those best qualified to explain it to us. I think Miss Eyles has done an excellent piece of work in bringing together the many intangibles that form the basis of our present knowledge of how fusion is attained, and it is only by a sound knowledge of the physiology of vision and the associated neuron-anatomy that we can understand how fusion is made possible or impossible.

We must try to assign the most exact etiologic factor possible to each case of squint for if we are able, by a good history and a thorough examination, to find the etiology, we are well on the way to a complete physiologic cure.

True, some etiologic factors cannot be found and others cannot be removed and remain as permanent barriers. Still other barriers may be removed only after diligence and perseverance in the best of teaching techniques.

How diligent can you get? Certainly training should go on until all removable barriers are removed and the teaching phase given an intensive trial. For orthoptics becomes a teaching process only when there are two good eyes that are reasonably matched one to the other and an I.Q. high enough to learn new skills or relearn neglected skills.

As Miss Eyles has mentioned, if we assume there is a fusion center at some cortical or subcortical level we make everything very simple. Then, in the absence of a recognizable physical defect or recognizable functional defect, we can place failures into a comfortable fusion-defect group, dismiss them and work on the easy ones.

In the early days of orthoptics this group was a very large one, but it is dwindling in the face of better teachers and better techniques. I do not think the group will entirely disappear, but it will no longer be the wastebasket to catch all the failures from either side of the machine.

Most of the fusion-difficulty cases in those acceptable for training seem to occur in that group of alternators who had an early onset of squint—that is some time before the age of two—and who have been neglected. Many have had absolutely no fusion experience and have to be taught everything artificially. Their barriers to fusion are a fixed angle of squint, anomalous correspondence, deep alternating suppression, and complete comfort in their monocular world. The deviation should be corrected surgically in this group as early as possible, even before the child is capable of performing some of the tests you would like to do. Nothing is gained by waiting until he is capable of being tested for the deviation will have to be corrected in any case. The child is incapable of straightening his eyes—a mechanical barrier—even though he learns skills all the way up the binocular ladder.

If such an alternator is first seen at the age of 6 or 7 years, then preoperative training may be given to good effect if the angle is not so large as to make training all but impossible. In this latter case the last barrier to be removed will be the mechanical one of heterotropia and, needless to say, training should follow surgery as soon as possible and be as intensive as necessary to obtain binocular vision with fusion for casual seeing and reading.

Miss Eyles has pointed out that one small patient may harbor a whole nest of barriers to fusion, and it is fortunate that sometimes we can remove two barriers at once. Antisuppression exercises are the same, basically at least, as antianomalous exercises and what a surprise occasionally is met when the surgical correction of a heterotropia also abolishes the anomalous correspondence.

Were I to be given a single choice of the many aids to fusion I would choose a good technician.

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OPTIC ATROPHY IN DIABETES MELLITUS*

A REPORT OF THREE CASES IN ONE FAMILY

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One of the rarer ocular complications of diabetes mellitus is atrophy of the optic nerve. In a review of the literature, the precise pathogenesis of this phenomenon is infrequently discussed, the assumption being that the atrophy occurs following a retrobulbar neuritis and is, therefore, postinflammatory.

Duke-Elder states[1] that retrobulbar neuritis is a rare complication in diabetes, but by no means unknown. It attacks people at all ages, and the great majority of the victims are males. There is characteristically a sudden onset, with development of a central scotoma, which may clear or may persist indefinitely, resulting in some degree of permanent atrophy. As to the mode of origin of the optic neuritis, several theories are advanced. These include the existence of diabetic toxins; the rendering of the patient more prone to exogenous poisons, such as alcohol and tobacco; the possible etiologic factor of diabetes-induced arteriosclerosis; and, finally, some connection with diabetic acidosis, raising a theoretical parallelism between alcohol and acetone.

Actually, the type of optic atrophy occurring in diabetes mellitus is generally accepted as being secondary to an optic retrobulbar neuritis, the exact cause of which is poorly understood, but which is usually rather sudden and dramatic in onset, and which leaves the patient with more or less residual optic atrophy and loss of vision (usually central).

Rea[2] tabulates the ocular complications in 4,001 eyes of diabetics. Of this group, 27, or 0.65 percent, had an optic atrophy. In 914 eyes of nondiabetics, he found 4 cases, or 0.4 percent, of optic atrophy. These figures would bear out Duke-Elder's assertion that the condition of retrobulbar neuritis is a rare one in diabetes mellitus.

This paper is a report on three members of a family of nine siblings. These three each had severe, long-standing, and poorly controlled diabetes mellitus, and each had a slowly progressive primary optic atrophy. The other six children in the family, as well as the parents and grandparents, did not have diabetes and demonstrated no evidence of an ophthalmic disorder of any sort. In the histories of the three children afflicted jointly with diabetes and atrophy of the optic nerve, there is no hint of a clear-cut episode of optic neuritis, retrobulbar or otherwise, and the visual loss has been insidious and gradual in each instance.

It will be suggested that perhaps the relationship of diabetes to the optic atrophy in these cases is not necessarily contingent upon an actual "neuritis," (that is, an acute inflammatory reaction in the axones of the ganglion cells of the retina) with associated clinical symptoms and signs, but may instead be analogous to the degenerative process which occurs in tabes and taboparesis.

Although it is true that the pathologic process in neurosyphilis involving the optic nerve was once held to be a primary degeneration of the ganglion cells with consecutive atrophy of the nerve fibers, the consensus of opinion now favors a primary peripheral and interstitial neuritis (a "cirrhosis" involving the supportive tissue of the nerve), followed by an axone degeneration. In the strictest sense, therefore, the atrophy that occurs with tabes is secondary to a neuritis, retrobulbar in location. Clinically, however, this type of atrophy is readily

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distinguished from the less insidious types, and the ophthalmoscopic picture is given the term of "primary optic atrophy" because there is no residual evidence of inflammation. If a retrobulbar neuritis, of whatever cause, occurs in the trunk of the nerve, there will be no ophthalmoscopic findings and the end result will also be "primary" atrophy. When the lesion is near the nervehead, the edema of the disc is marked and leaves behind, in the ensuing atrophy, those residual ophthalmoscopic traces of hemorrhage, exudate, vascular abnormality, and blurring of the disc margins that give the picture the name of "secondary" or "consecutive" optic atrophy.

Review of literature

The literature is not replete with discussion and opinion regarding the optic atrophy which occurs in diabetes. In three pages devoted to a discussion of the association of diabetic neuropathy and retinopathy, Rundles[3] discusses the retinal findings in his series of 125 cases, but does not mention optic atrophy. Burch[4] states that of 292 diabetics, one had optic atrophy, but does not elaborate.

O'Brien and Allen[5] studied the fundi of 555 patients under 31 years of age with diabetes mellitus. They summarized the retinal findings and found that four percent of their series showed retinopathy, but did not mention optic atrophy. McKee,[6] in 1941, analyzed 2,360 cases of diabetes mellitus from the ophthalmic point of view. Of this group, 476 persons had pathologic conditions of the retina which he felt to be more or less definitely related to diabetes, but there was only one case of atrophy of the optic nerve listed, and he apparently did not definitely ascribe this to diabetes.

As a matter or fact, Folk and Soskin,[7] in a discussion of diabetic ophthalmoscopic findings and of the differentiation between the findings in diabetic, arteriosclerotic, and albuminuric retinitis, stated that one of the criteria for diabetic retinitis is a "normal or nearly normal disc." While this actually had reference to the fact that, in the other two conditions, waxiness or edema of the disc serves to distinguish them, it does point up the fact that optic atrophy is a rare occurrence, and one not commonly associated with diabetes.

A specific reference to retrobulbar neuritis is to be found in a discussion by Frances and Koenig,[8] written in 1926. They stated that "Retrobulbar neuritis arising in the course of diabetes, the evident result of an endogenous toxin or toxins liberated during the disease, which exercises a selective action on the papillo-macular bundle of the optic nerve, is undoubtedly a clinical entity."

These authors felt that the precise toxins which attack the papillomacular bundle were not known, but that the ketone bodies (beta-oxybutyric acid, aceto-acetic acid, and especially acetone, along with some unidentified organic acids) might be the cause, and that the resultant amblyopia usually disappeared in from 3 to 8 months, the amelioration of the symptoms showing a definite relation to the disappearance of the ketone bodies from the urine.

Rönne is said to have described the pathologic process in the papillomacular bundle at necropsy of two patients, whom he had also studied clinically, as being a degenerative one, rather than a neuritis, and similar to that found in alcohol and tobacco amblyopia except for the presence, in diabetes, of "Kornchen cells," which are not found in the others.

Frances and Koenig concluded, after a case report, that glycosuric amblyopia, involving the central visual fields, the result of an endogenous toxin which is probably acetone, is a clinical entity.

Case reports

This recognized disease complex does not apply, however, to the three patients to be discussed herein, since the onset in their

cases was insidious rather than sudden, the absolute scotoma is peripheral rather than central, and the condition has not improved with better control of the diabetes, but rather has steadily grown worse (in one patient over an 11-year period).

CASE 1

History. I. J. T., a young woman, aged 21 years, was admitted to Parkland Hospital on January 26, 1947. This patient had been a known diabetic since the age of four years, but the onset of her visual difficulties occurred at the age of 12 years, when she found that she could not read fine print. By the time she was 17 years of age, she was unable to read school books; however, she was graduated from high school. At present she is a domestic. She states that she has little difficulty in performing housework but that she does not read any but the largest print and frequently stumbles and runs into objects.

Her past medical history, except for the severe diabetes mellitus, which has never been well regulated with insulin and diet, and the progressive visual difficulties, is essentially negative.

The family history is entirely devoid (with the exception of the two younger siblings) of diabetes or of significant visual disturbance. The grandparents were not examined, but there is no history suggestive of a hereditary type of optic atrophy. The parents do not have diabetes mellitus and the father, who is no known relation to the mother, is serologically negative with no history of treated lues. His visual apparatus, as well as his carbohydrate-metabolizing system, are normal. The mother was not able to report for examination, but she is not a diabetic and a recent communication states that an ophthalmic examination is essentially normal.

Eye examination. I. J. T. stated that her vision seemed to be appreciably worse after an attack of measles in 1941. The onset of visual disturbance was considerably before this, in 1936, at the age of 10 years. She stated her vision was better at night than during the day. Physical examination of this patient was essentially negative except for the eye findings. The pupils were round and regular (2.5 mm.) and reacted very sluggishly to light and accommodation, with no consensual reaction.

Vision was: R.E., 15/200, JO; L.E., 20/200, JO. Cycloplegic retinoscopy showed a refractive error of: R.E., −0.75D. sph. ⌒ +1.0D. cyl. ax. 180°; L.E., −1.0D. sph. ⌒ +1.0D. cyl. ax. 180°. The patient subjectively accepted: R.E., −1.0D. sph. ⌒ +1.0D. cyl. ax. 180°; L.E., −0.5D. sph. ⌒ +0.5D. cyl. ax. 180°, which brought the vision only to: R.E., 20/200, JO; L.E., 20/200, JO; hardly an appreciable improvement.

Funduscopic examination revealed the media to be clear. The peripheral fundus was normal with no hemorrhages, exudates, or vascular abnormality. Both optic discs were grayish-white, centrally, fading to white peripherally. The right eye demonstrated more pronounced pallor than the left. Both discs showed definite loss of substance. The disc margins were clear-cut and sharply demarcated (fig. 1).

Visual-field studies with a 5-mm. white target at 330 mm. revealed bilateral, quite regular constriction of from 10 to 30 degrees, somewhat more pronounced in the right eye, especially temporally (fig. a). Attempts to obtain color fields were not satisfactory, and it was found that the patient was red-green color blind, and recognized a solid-blue object with difficulty.

The blindspots with a 5-mm. target at one meter were elongated in a vertical direction. There were no isolated scotomas, and it was felt that the visual fields were not pathognomonic of any particular disease entity. The responses with a target smaller than 5 mm. were not conclusive. The intraocular pressure was: R.E., 18 mm. Hg (Schiøtz);

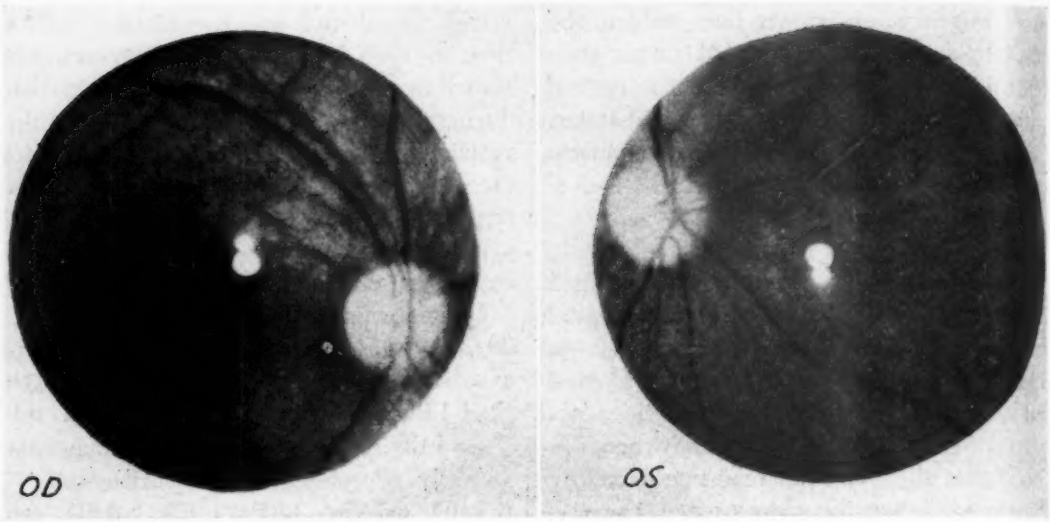

Fig. 1 (Stansbury). Fundi of *Case 1,* showing pallor as well as sharp outline of optic discs. Vision is: 20/200, JO, O.U. The maculas normal.

L.E., 18 mm. General physical and neurologic physical examinations were entirely normal.

Laboratory reports. Blood studies revealed: Hb., 13.5 gm.; R.B.C., 4.21, W.B.C., 7,150, with normal differential. Urinalysis, sp. gr., 1.013; acid; sugar, 4 plus; negative acetone. The blood serology was negative; the fasting blood sugar, 277. Spinal fluid examination showed normal pressure, no cells. Wassermann and colloidal gold tests were negative. X-ray films of the optic foramina showed no constriction nor abnormality and the sella turcica was normal.

This patient was regulated on 40U protamine zinc insulin and 15U regular insulin before breakfast and 10U regular insulin before supper, and was discharged without

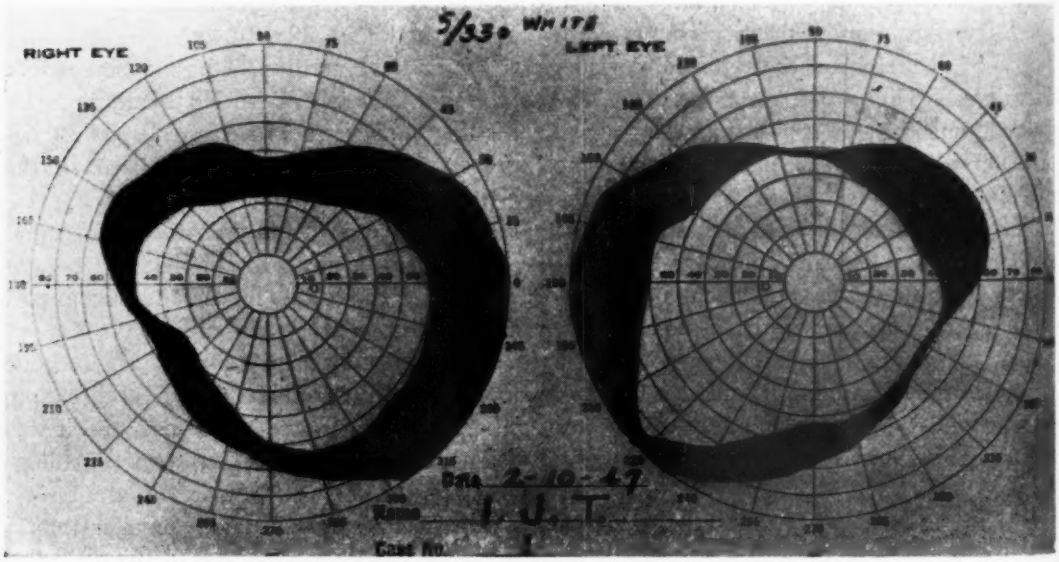

Fig. a (Stansbury). Peripheral fields of *Case 1,* 5/330 white.

other treatment. Intensive vitamin-B therapy had been employed since 1944, and it was advised that this be continued.

CASE 2

History. D. L. T., a girl, aged 14 years, was a sister of I. J. T. She had been a known diabetic since the age of seven years and was admitted to the hospital on February 4, 1947. This patient, a high-school sophomore, made excellent grades in her school work until one year ago, when she began to have trouble reading her lessons.

The patient's past medical history began with the sudden onset of loss of weight at the age of seven years, whereupon her parents diagnosed diabetes mellitus (having had previous experience with the first patient), and arbitrarily placed her on insulin and a diabetic diet. There was no history of diabetic coma, but there had been frequent episodes of mild insulin reaction. She had had diphtheria, measles, and mumps in childhood, and there was a history of recurrent furunculosis. Two years prior to admission, at the age of 12 years, she first noticed diminution of vision, particularly for near, with blurring of print and lacrimation. There was no complaint of difficulty with vision at night. Four weeks before her admission, she had been hospitalized elsewhere with left lobar pneumonia and left-sided pleurisy.

General physical examination revealed a well-nourished, prepuberty white girl, with no positive findings except for an auscultatory friction rub at the left 5th rib posteriorly. Neurologic examination was entirely normal.

Eye examination. Vision was reduced to: R.E., 20/200, J2; L.E., 20/200, J4. The pupils measured 4.5 mm., and were round and equal, with normal light and accommodative, as well as consensual, reactions. The media were clear, and the peripheral fundus, macula, and vascular tree were normal binocularly. The optic discs were flat and sharply demarcated, with a peripheral white ring fading to gray centrally, and had the typical appearance of a developing primary optic atrophy (fig. 2).

Retinoscopy with homatropine cycloplegia showed: R.E., −0.75D. sph., L.E., −0.50D. sph., but subjectively with: R.E., −0.75D. sph.; L.E., −0.25D. sph., vision remained only very slightly improved at: R.E., 20/200 J2; L.E., 20/200, J3.

The color vision was defective with the American Optical color charts, but the patient was able to distinguish and recognize red, green, and blue objects. The intraocular pressure was: R.E., 21 mm. Hg (Schiøtz); L.E., 25 mm. Visual fields (fig. b) showed moderate peripheral constriction, more marked temporally, for 5/300 white. Tangent-screen studies revealed only moderate enlargement of the blindspots, more marked on the right. The color-fields could not be examined satisfactorily due to the defective color-vision, but in general the form-fields with 5-mm. targets were larger for blue than for red and green, which was felt to corroborate the existence of neuroretinal rather than choroidal damage.

Laboratory studies showed the serology to be normal (Kline and Mazzini). C.B.C.: Hb., 10 gm., R.B.C., 3.8; W.B.C., 9,100; normal differential. Urine: sugar, 3 plus; 1.025, sp. gr.; acid reaction; negative acetone. Fasting blood-sugar, 190. The spinal fluid examination on February 5, 1947, revealed a normal pressure, but 3 segmented cells and 128 lymphocytes; on February 7th, two days later, the spinal fluid showed no segmented cells and 4 lymphocytes. The spinal fluid Wassermann and colloidal gold tests were negative. X-ray studies of the optic foramina and sella turcica were normal.

During her hospital stay, the patient developed an acute pleuritis and some parenchymal pulmonary consolidation which was thought to be an exacerbation of the previous episode and unrelated to her diabetes. She was felt to be adequately controlled at

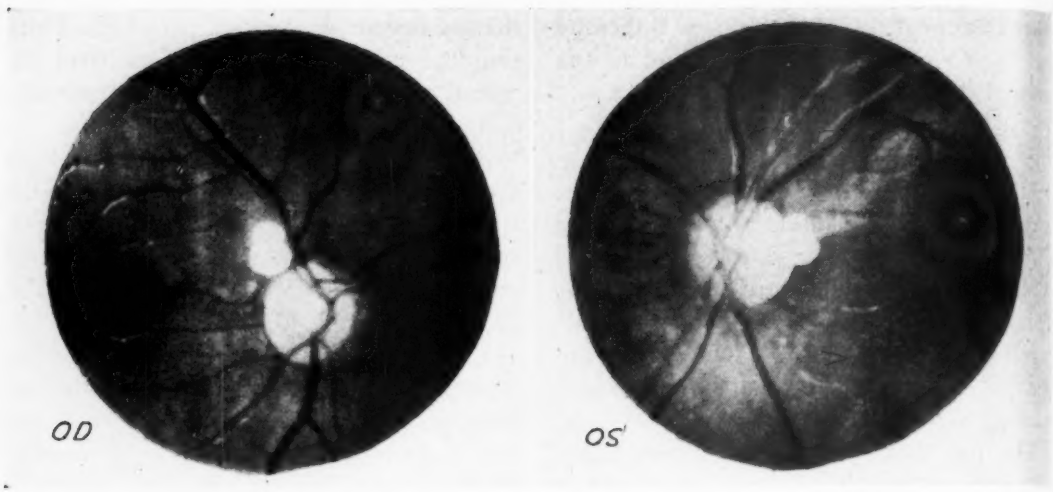

Fig. 2 (Stansbury). Fundi of *Case 2.* Vision is: R.E., 20/200, J2; L.E., 20/200, J3.

the time of her discharge on protamine-zinc insulin (40U) and regular insulin (15U) before breakfast, and regular insulin (10U) before the evening meal, in addition to a 1,390 calorie diet. On this regimen, the urine-sugar varied from 3 plus to a trace, with no acetone, and it was not felt possible to obtain better control. (Note: Information has been received that this patient has developed tuberculosis of the spine since this hospitalization.)

CASE 3

History. R. T., a boy, aged 13 years, was admitted to the hospital on February 4, 1947. He was a brother of patients reported in Case 1 and Case 2. The boy had been known to have diabetes since he was seven years of age when the mother tested his urine, found sugar, and arbitrarily placed him on insulin. He had never been taken to a physician for regulation and stated that he frequently experienced mild insulin reactions which were

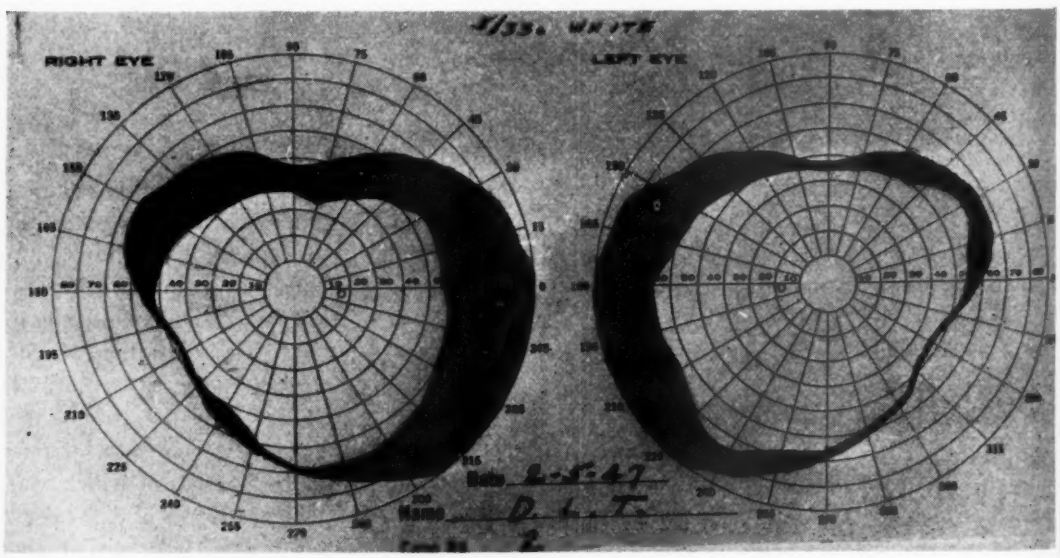

Fig. b (Stansbury). Peripheral fields of *Case 2.*

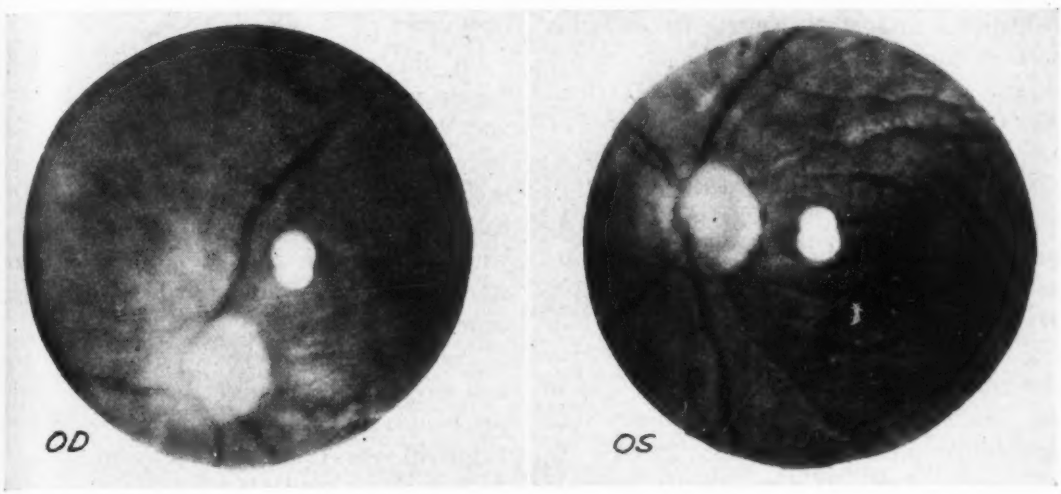

Fig. 3 (Stansbury). Fundi of *Case 3.* Vision is: R.E., 20/200, J6; L.E., 20/100, J6. The haziness of the disc margin on the right is an artifact.

relieved by drinking fruit juice or eating a piece of candy. Two years before admission, at the age of 11 years, he noticed the inability to read for prolonged periods without blurring of the print, and although in the seventh grade and not failing in his studies, he was conscious of slowly diminishing visual acuity. There was no difficulty with vision at night.

Eye examination. The past medical history was noncontributory, and general physical examination, with emphasis on the neurologic aspect, was entirely negative except for the ocular findings. The boy had not reached puberty. Vision was: R.E., 20/200, J6; L.E., 20/200, J6. Homatropine retinoscopy showed: R.E., +0.50D. cyl. ax. 90°; L.E., +0.25D. cyl. ax. 100°; but subjectively he accepted: R.E., plano; L.E., +0.25D. cyl. ax. 90° to make his best corrected vision: R.E., 20/200, J6; L.E., 20/100, J6. The pupils were round and equal (4 mm.), reacted to light and accom-

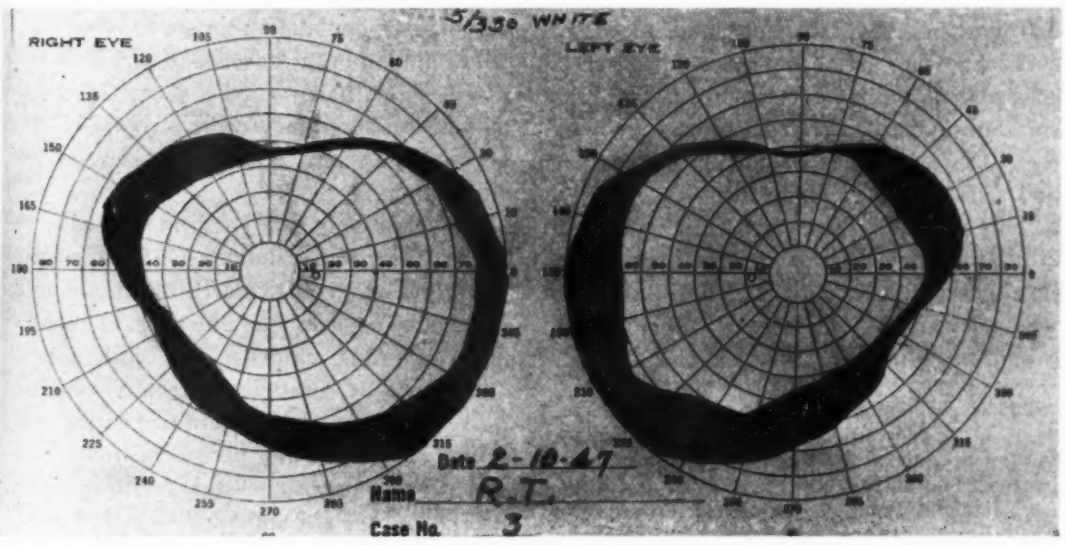

Fig. c (Stansbury). Peripheral fields of *Case 3.*

modation, and the consensual reaction seemed sluggish. The extraocular movements were normal, and the media clear. The peripheral fundi showed no evidence of hemorrhage, exudate, or vascular abnormality, but the appearance of the optic discs was quite similar to that of the other two patients. The margins were sharply demarcated, the physiologic cups wide, the periphery pale white (especially temporally) fading to gray centrally (fig. 3). One observer described debatable degenerative changes in the macular regions of this patient. The intraocular pressure was: R.E., 25 mm. Hg (Schiøtz); L.E., 27 mm. Visual fields in this case, too, showed nothing pathognomonic, but rather a concentric peripheral constriction, more marked temporally (fig. c). The color vision was defective, and attempts to outline the color fields were not successful. The blindspots of Mariotte were elongated vertically, more so on the right, but there were no central scotomas.

Laboratory studies revealed a negative serology. C.B.C.: Hb., 14; W.B.C., 6,700; normal differential. Urine: sugar, 2 plus; sp. gr., 1.025; acid; acetone negative. Fasting blood-sugar, 305. Spinal fluid on February 5, 1947, was clear; the pressure was normal, but there were 3 segmented cells and 120 lymphocytes; on February 15, 1947, a repeat examination revealed 3 segmented cells and no lymphocytes; spinal fluid Wassermann and colloidal gold tests were negative.* X-ray studies of the optic foramina and sella turcica were normal.

This patient's diabetes was regulated with 20U protamine-zinc insulin and 15U regular insulin before breakfast. His hospital course was uneventful, and he was discharged on a high-protein, high-vitamin diet.

* The spinal fluid findings in Case 2 and Case 3 were felt to represent a possible error in that both specimens with a high cell-count were collected by the same student on the same day, and in a later specimen, each patient showed a nearly negative cell-count. It must be admitted that the significance of the spinal fluid findings is not clear.

Comment

Of the remaining six children in this family whose ages were 23, 20, 17, 15, 11, and 9 years, respectively, the three oldest were available for examination. Vision in each of these siblings was: 20/20 J1, O.U., color vision was normal, and the optic nerves were normal to examination. In fact, there was no evidence of an ophthalmic pathologic condition or diabetes.

The father, aged 46 years, was examined and was found to be normal except for early presbyopia; the mother of these children (aged 40 years) and the three younger children were not available for examination, but there were no visual complaints, and all had been tested for diabetes and found to be free of signs or symptoms.

As has been stated, there was no known hereditary visual affection and no history of diabetes in the grandparents or the more remote ancestors. Generally speaking, this large family, with the exception of these three diabetic children, seems to be normal physically, and of sturdy stock. The father's occupation was that of a farmer.

Discussion

In considering the possibility that a causative factor other than diabetes might be responsible for the optic atrophy in these children, and in the absence of any indication that syphilis had played a part, the disease pattern of hereditary optic atrophy or "Leber's Syndrome" was given serious consideration.

This relatively rare condition, of unknown etiology but with a definite "hereditary tendency, affecting particularly males about the 20th year of life, . . . showing in its early stages fleeting evidence of optic neuritis, followed by a secondary atrophy which involves a serious impairment of central vision with retention of the peripheral fields"[9] . . . has several characteristic features. These are listed by Raaf and Bair[10] as: (1) A predilection for males; (2) average age of on-

set 23 years for males and 25 years for females (among Europeans); (3) rapid impairment of vision, with maximum disability within two months from the onset, and rarely with advancement of symptoms after six months; (4) patients are usually in good health, no other disease being characteristically associated with hereditary optic atrophy; (5) the sole complaint is usually diminution of vision, with the most common defect being a central scotoma, although peripheral defects are frequently seen; (6) color blindness is usually the only other symptom; (7) in early stages, the fundus may be normal or may show typical optic neuritis, optic atrophy supervenes, and the disc never regains its normal appearance; (8) from 10 to 29 percent of all patients show some subsequent improvement and, if the age of onset is early, the prognosis is better; furthermore, there is an inherited tendency to improvement; (9) in 93.6 percent of cases (European series) the disease was transmitted by the mother.

It will readily be seen that the majority of these criteria are not met by the three subjects of this paper.

Fisher,[11] in 1916, advanced the idea that Leber's optic atrophy is the result of hereditary changes in the pituitary gland, probably temporary in character, but clinical and X-ray studies have failed to bear this out, and the theory which blames irregularities in the growth of the sphenoid bone has not received much support. In the patients under discussion, there was no evidence of a primary pituitary disturbance, and the films of the skull did not reveal prominent clinoid processes or deep sella turcicas.

Pathologically, there is said to be[12] atrophy of the ganglion cells and nerve-fiber layer of the retina in Leber's disease, with the atrophy of the nerve limited to the papillomacular bundle and other parts of the nerve normal. There is destruction of the medullary sheaths but no inflammatory signs (this report records the autopsy findings in but one case), which leads its author to conclude that the underlying pathologic process is a primary neuronic (inherited) degeneration localized in the optic nerve, rather than a toxic retrobulbar neuritis secondary to neighboring sepsis and infected tissue which bears an inherent vulnerability.

Bhaduri[13] suggests that the condition may be embryologic, with a common germ-plasm defect in the anlage of the retina and the inner ear. In substance, however, it would seem that, by virtue of their failing to meet practically all of the criteria for hereditary optic atrophy, this syndrome can be excluded as a possible diagnosis in the patients herein reported.

In a further consideration of the differential diagnosis of these cases, any of the more remote causes for primary atrophy of the optic nerve must be considered. As has been stated, primary optic atrophy occurs subsequent to retrobulbar neuritis, when the inflammation confines itself to the trunk of the nerve and does not approach the nerve-head.

Duke-Elder[14] considers among the causes for retrobulbar neuritis followed by optic atrophy, the group of so-called "neurotrophic virus infections." These include disseminated (multiple) sclerosis, acute disseminated encephalomyelitis, neuromyelitis optica (of Devic), diffuse periaxial encephalitis (of Schilder), and herpes zoster. All of these are characterized by an extensive demyelinization of the optic-nerve fibers where these fibers are myelinated (hence retrobulbar), followed by degeneration of the axis cylinders, and finally a proliferative gliosis. In the subjects under discussion, the absence of positive neurologic signs, except ophthalmologically, along with the absence of clinical history and peripheral nerve damage, would appear to exclude this group of diseases.

In an exhaustive study concerning the relationship between Recklinghausen's disease (or multiple neurofibromatosis) and

gliomatous tumors of the optic nerve, Davis[15] makes the statement that many cases of unexplained atrophy of the optic nerve may be due to early tumors of the nerve, some of which may have been incorrectly diagnosed as retrobulbar neuritis.

Exophthalmos need not be present, but the diagnostic leads that are of value in establishing the presence of atypical neoplasms of the nerve are said to be subcutaneous nodules which are manifestations of the general condition, café au lait pigment patches on the skin, and certain typical changes, visible by X ray, involving the sella turcica and the optic foramina.

None of the three patients herein studied showed evidence of subcutaneous tumors, café au lait spots, skull changes visible to X ray, exophthalmos, or evidence of increased intracranial pressure, so that the possibility of tumor of the optic nerve being responsible for their optic atrophy seems remote.

These children were all three well nourished. The two younger siblings might be said to be somewhat retarded as regards pubertal changes. None of them showed evidence of an avitaminosis which might have produced an optic neuritis of the axial type. Since the onset of the visual disturbance, although occurring at about the same age in each patient, did not occur in the same calendar year, or coincident with an epidemic such as typhoid, influenza, and so forth, the possibility of an optic neuritis from endogenous toxins (other than diabetic) seems remote. External toxins, such as tobacco, alcohol, lead, arsenic and so on, were considered and then rejected as highly improbable due to the complete lack of contact. There was no evidence of adjacent infection, periorbital, intracranial, or in the paranasal sinuses, to produce optic neuritis and subsequent atrophy. As a matter of fact, two of the cardinal signs of optic neuritis were absent; namely, sudden onset and central scotomas. On the contrary, the progress had been very insidious and, although central vision was defective, the absolute scotoma was peripheral in each case.

It was suggested that, since each of these children had had frequent episodes of insulin shock, this alone might in some manner produce atrophy of the optic nerve. However, it was felt that, if this relationship existed, such a syndrome would be recognized and established.

Summary

To summarize, then, three children of a farming family of nine siblings have been shown to have had rather severe diabetes mellitus from early childhood and, in addition, each of these children has a pathologic condition of the eyes manifested by an absolute peripheral scotoma, a relative central scotoma, defective color vision, and a well-advanced primary optic atrophy (binocular), but no evidence of diabetic retinopathy.

The other six children in the family, as well as the parents and grandparents, are free of diabetes, and none shows evidence of optic atrophy or other ocular disease. The evidence may be circumstantial on these grounds, but from the standpoint of the selectivity of the optic atrophy, it would seem that, in these patients, there is a direct relationship between their diabetes and their visual symptoms.

A consideration of the possible etiology of the atrophy of the nerve, other than following a retrobulbar neuritis secondary to endogenous diabetic toxins, gives no clues, and an attempt has been made to rule out the common causes for retrobulbar neuritis and optic atrophy, including lues, exogenous toxins, the virus neuropathies, endocrine dysfunction, cranial malformation, tumor of the optic nerve, and particularly Leber's syndrome of hereditary optic atrophy. The weight of evidence in these patients seems to point to diabetes as the factor responsible for their eye-findings, but the pertinent history, as well as the clinical findings,

differs rather markedly from the usual case of this type in that there is no clear-cut episode of retrobulbar neuritis and the loss of central vision has been relative and gradual, the papillomacular bundle being only incidentally involved.

It is suggested that perhaps the underlying pathologic process in this group of patients is indeed a degenerative process—rather than an actual inflammatory "neuritis"—of the type that is said to occur in alcohol or tobacco amblyopia, but differing rather markedly in that the process here does not show a predilection for the papillomacular bundle, but rather involves the entire bundle of nerve fibers somewhere behind the globe and anterior to the external geniculate bodies. Furthermore, the process appears to be a particularly insidious one. Alternately, this might represent a "tabes-like" type of quiet neuritis and axone-degeneration.

In either case, the causative agent is not known, but it can be speculated that some type of endogenous poison, whether ketone-bodies or something else, occurring in the presence of active diabetes mellitus, is responsible for the condition. That there may be another, as yet unknown and undetermined, factor in this syndrome of optic atrophy and diabetes is a possibility. It seems reasonable to suppose that a joint defect in the germ plasm—some type of aberration common to the anlage of the pancreas and the visual organ—may be basically responsible for the presence of the two conditions of diabetes and optic atrophy in these patients, and this relationship could conceivably apply wherever these diseases coexist.

Conclusion

Optic atrophy can occur as a complication of diabetes mellitus. It is probably preceded by some type of retrobulbar neuritis, but it is suggested that in certain instances, the process may be primarily a degenerative one.

4105 Live Oak Street (4).

References

1. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, C. V. Mosby Co., 1941, v. 3, p. 3005.
2. Rea, R. L.: Neuro-Ophthalmology. St. Louis, C. V. Mosby Co., 1938, p. 474.
3. Rundles: Diabetic neuropathy. Medicine, **24**:160 (May) 1945.
4. Burch: Ocular conditions associated with diabetes. Lancet, **48**:137 (Apr.) 1928.
5. O'Brien and Allen: Ocular changes in young diabetic patients. J.A.M.A., **120**:190, 1942.
6. McKee: Fundus oculi in diabetic patients. Arch. Ophth., **25**:773 (May) 1941.
7. Folk, M. L. and Soskin, S.: The fundus oculi in diabetes mellitus. Am. J. Ophth., **18**:432, 1935.
8. Frances and Koenig: Retrobulbar neuritis in diabetes. J.A.M.A., **87**:1373 (Oct.) 1926.
9. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, C. V. Mosby Co., 1941, v. 3, p. 2998.
10. Raaf and Bair: Leber's disease. Am. J. Ophth., **21**:384, 1938.
11. Fisher, J. H.: Leber's disease (hereditary optic atrophy): A suggestion as to its cause. Tr. Ophth. Soc. U. Kingdom, **36**:298, 1916.
12. Alexander: Leber's disease: Report of four cases in one family. Texas State J. Med., **39**:301 (Sept.) 1943.
13. Bhaduri: Hereditary optic atrophy (Leber's disease). Calcutta M. J., **42**:1 (Jan.) 1945.
14. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, C. V. Mosby Co., 1941, v. 3, p. 2089.
15. Davis, F. A.: Primary tumors of the optic nerve: A phenomenon of Recklinghausen's Disease. Arch. Ophth., **23**:735; 957, 1940.

NOTES, CASES, INSTRUMENTS

AN OPERATION FOR SHORTENING A RECTUS MUSCLE

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Marquette, Michigan

Operations for increasing the action of an eye muscle by advancement or resection are attended by the possibility of the sutures giving way and decreasing the amount of correction. This is especially true when the muscle is attenuated or stripped of its fascial covering.

In the Reese resection the pull of the sutures is in the direction of the muscle fibers making it easy for the sutures to slip toward the cut end. This is most likely to occur when tension is placed on the suture to draw the muscle into place against the stump before tying. It can still occur after the ends are in position and the sutures tied but is less apt to at this time, as some of the pull is now in a horizontal direction across the muscle fibers.

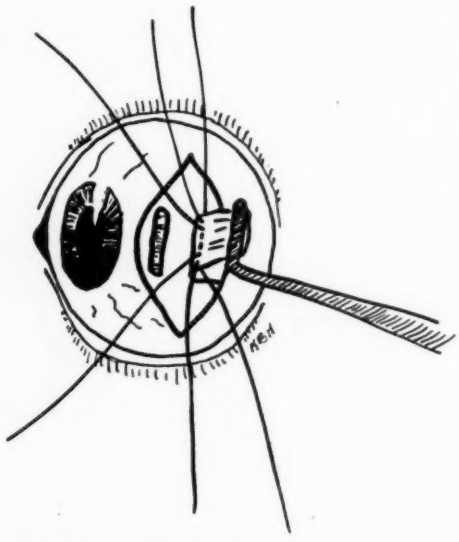


Fig. 1 (Hornbogen). The central mattress suture has the loop on the outside instead of the under surface of the muscle, and the conjunctiva is not included.

The modification here presented is one in which the pull of the suture is always across the muscle fibers and not with them. The muscle is exposed in the usual manner but not stripped of its fascial covering. A muscle forceps is applied near the insertion and the tendon severed leaving a stump of about 1.5 mm.

A central mattress and two wing sutures are placed in the muscle at the desired distance, as in a Reese operation, except that the central mattress suture has the loop on the outside instead of the under surface of the muscle, and the conjuntiva is not included (fig. 1). They are next passed through the stump. At no time during this stage should any pull by the sutures be exerted against the fibers. As tension is placed on the central suture to draw the muscle and the stump together, the assistant holding the forcep attached to the end of the muscle, draws it toward the canthus making a V with the sutures at the apex (fig. 2). This puts all of the suture pressure across the fibers. The suture has such a firm hold on the muscle that it is not neces-

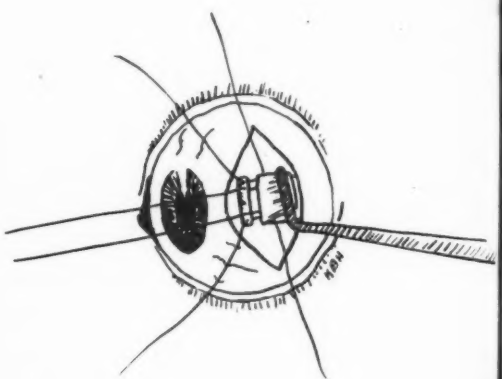


Fig. 2 (Hornbogen). As tension is placed on the central suture, it is drawn toward the canthus, making a V with the sutures at the apex.

ary to rotate the globe to bring the stump to e muscle.

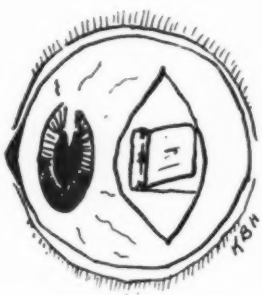
The amount of force applied need be gov- ned only by the breaking strength of the ture or by the amount necessary to tear rough the bite of stump and episclera. he flap is held in this position until the tures are tied and cut.

The forceps are removed leaving a flap tendon beyond the sutured stump (fig.). This is now laid back, flat, along the uscle as far as it will reach and sutured it at each edge. If the amount of shorten- g is large and the excess tendon is unduly ng, a portion may be excised before it is tured in position (fig. 4). The conjunctiva closed separately. If one does not wish bury the knot of the mattress suture, it n be carried through the limbal flap of njunctiva and tied outside.

The suture material used can be that pre- rred by the individual operator. I have been using 4-0, 10-day, chromic gut for the mattress suture and 4-0 plain gut for the remainder.

This method is applicable to either the internal or external rectus. The thickening produced by the double layer of muscle is minimal and of no consequence and is not comparable to that produced by a tucking or a cinch operation. With this technique it is possible to mobilize the eyes earlier in the postoperative period.

401 Savings Bank Building.



g. 3 (Hornbogen). The forceps are removed aving a flap of tendon beyond the sutured stump.

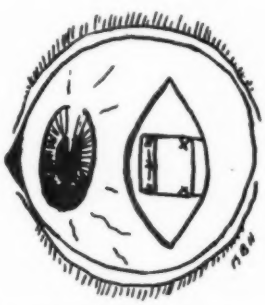


Fig. 4 (Hornbogen). If the amount of shorten- g is large and the excess tendon is unduly long, a rtion may be excised before it is sutured in posi- n.

BIPRONGED FORCEPS

AN INSTRUMENT FOR PLASTIC SURGERY

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In performing plastic procedures of the eyelids, it is frequently necessary for an assistant to apply forceps to areas immediately contiguous to that held by the surgeon. This is in order to prevent distortion in

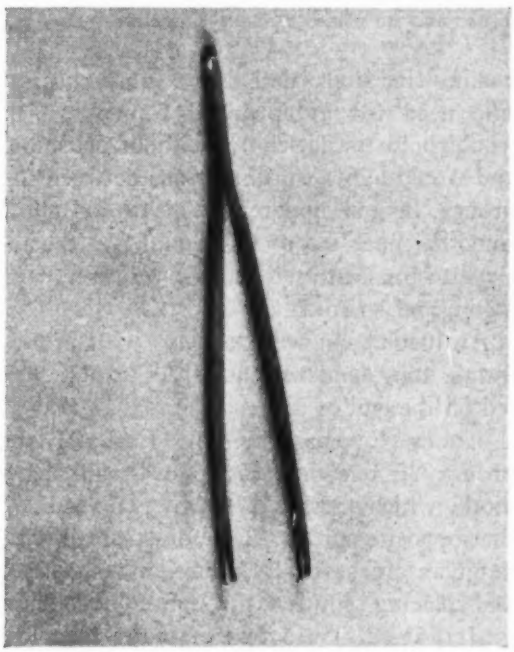

Fig. 1 (Callahan). Bipronged forceps for use in plastic eyelid surgery.

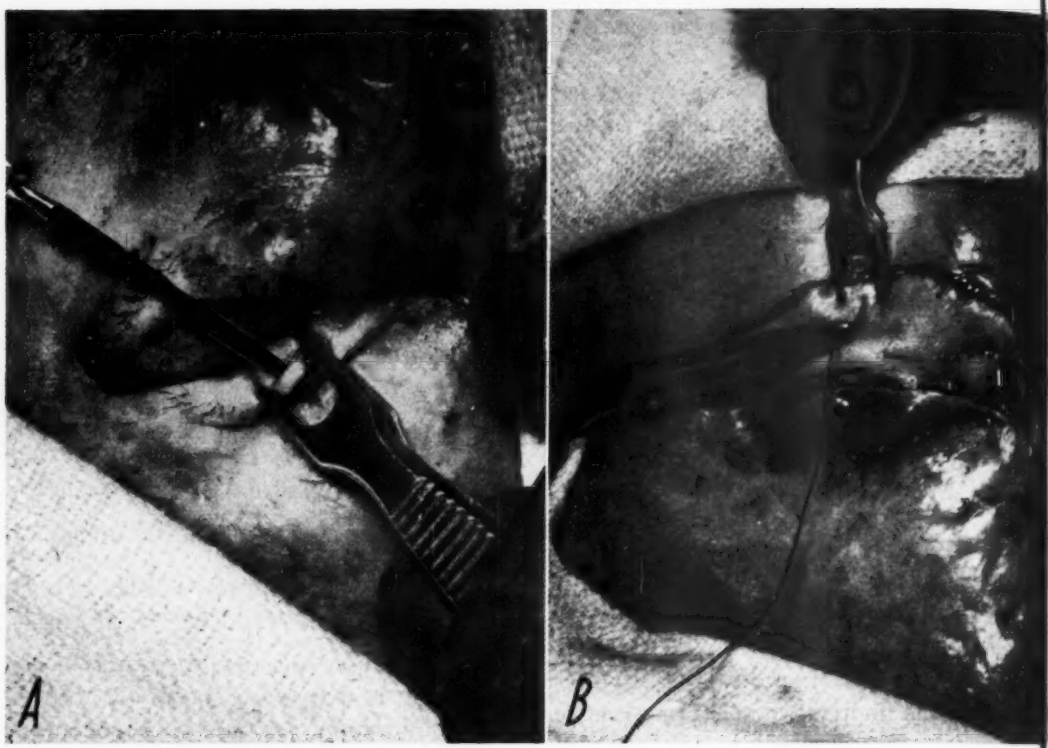

Fig. 2 (Callahan). (A) The bipronged forceps are holding the margin of the lower lid firmly, as Graefe knife denudes a small rectangular area of transitional epithelium. (B) A free-skin graft is place in position for the correction of ectropion. The bipronged forceps are holding the graft so that the fir suture may be placed without difficulty.

making incisions and in placing sutures. The need for a bipronged forceps which would hold tissue firmly, without crushing, and would allow working space between the prongs, became apparent. This would allow smoother handling of tissue and free the assistant for cutting sutures, sponging of blood, and so forth.

An instrument was designed with prongs 8 mm. long, and the interspace 5 mm., fitted on the end of a handle similar to that of a Lester forceps (fig. 1). Each of the prongs on one side carries a small sharp tooth, which meshes between two teeth on the opposite prong. Denuding small rectangular areas for lid adhesions (fig. 2A) and placing sutures through a free skin graft (fig. 2B) are two of many technical procedures facilitated by its use.

Medical College of Alabama (5).

FLEXIBLE CABLE SLIT CONTROL FOR BIOMICROSCOPE

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Steubenville, Ohio

It has long been felt that control of th width of the slitlamp beam should be s facilitated mechanically that the biomicros copist could alter the beam with utmos ease while examining pathologic details.

Of extant models of biomicroscopes, th Haag-Streit (Goldman),* and the Zeis (Comberg)† are examples of geared con trol of the slit width. The simplified Uni versal model biomicroscope‡ lends itself t

* Goldmann biomicroscope (Haag-Streit, Bern Switzerland).

† Comberg biomicroscope (Carl Zeiss, Jena, Ge many).

‡ Universal biomicroscope (Bausch & Lom Optical Co., Rochester, N.Y.).

incorporation of a flexible cable and knurled knob which will permit accurate, speedy slit-width readjustment without the fumbling in the dark which heretofore has inhibited alteration of the beam.

It is well known that the best results in visualizing the manifold optical changes of the ocular media are obtained with a combination of changing microscope focus, beam width, oscillatory field (Koeppe),[§] and panoramic sweeping of the beam across the field while employing the various modes of illumination. This type of optical scrutinization or "visual palpation" leads to definitive interpretation of the various refractive, reflective, and dispersive changes which characterize pathologic alteration of the ocular structures or media.

Direct focal illumination produces the finest optical delineation but unless the observer employs[‖] reciprocally the wide and narrow beam for observation and localization, he loses the full potentiality of biomicroscopy. The study of shadow formations ("optic reduplications"), vacuoles and bullae, deposits, and so forth, is enhanced by smooth control. Differentiation of extrasectional as well as sectional features in localization is helped by being able to widen and narrow the beam facilely. If students of the technique would learn at the outset to examine the eye with the various methods of illumination and with rapid beam-width control, considerable improvement in biomicroscopic technique would ensue.

Study of zonal discontinuity in the lens is benefited with ready control of the beam flux. After a survey of the lens with a wide beam, the transition to optic section for localization of details is smoother and more rapid in contradistinction to the present method of reaching to the slit housing for the slit-control screw and then twisting it to obtain the requisite narrowing. It is small wonder that the busy practitioner, unconcerned with the minutiae of biomicroscopy, may allow the slit to rust into fixation at one wide width. To help him as well as beginning students, I offer this suggestion for modification of a domestic biomicroscope.

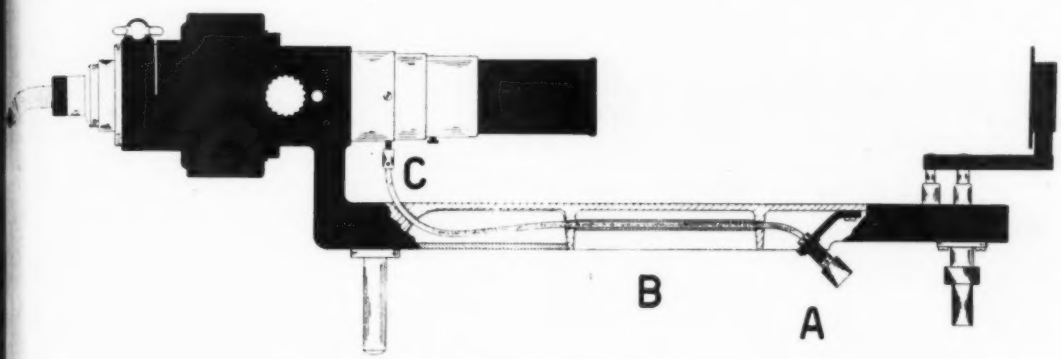

Fig. 1 (Stein). Cross-sectional view of apparatus.

The knurled knob is placed (fig. 1-A) at the distal extremity of the illuminating arm in proximal juxtaposition to the Arruga screw at such an angle as to be readily available to the controlling fingers of either the right or left hand. By means of a flexible cable affixed within the matrix of the illuminating arm (fig. 1-B), rotation of the knurled knob results in corresponding rotation of the inverted slit-control screw to which the cable is fastened (fig. 1-C), leading to alteration of the width of the corneal parallelepiped, lens prism, and optic section.

§ Koeppe: Cited by Berliner, pp. 69 and 108.

‖ Berliner, M. L.: Biomicroscopy of the Eye, 1943, v. 1, p. 79.

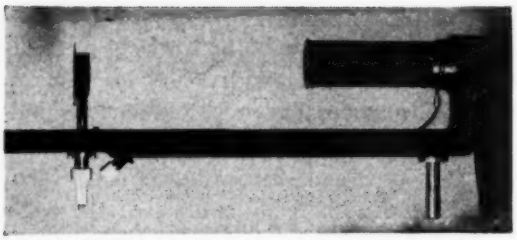

Fig. 2 (Stein). Universal model (Bausch & Lomb) biomicroscope with slit-control cable attached.

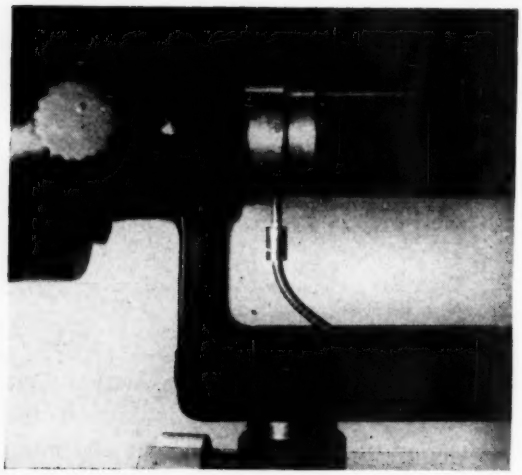

Fig. 3 (Stein). Close-up view of attachment of flexible cable to inverted slit screw.

Fig. 4 (Stein). Close-up view of distal control knob placed behind Arruga screw.

The photographs (figs. 2, 3 and 4) depict the appearance of the illuminating arm of the Universal (Bausch & Lomb) biomicroscope, modified according to the suggestion of the writer.

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TOTAL DISAPPEARANCE OF THE IRIS DUE TO ESSENTIAL ATROPHY*

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Apparently all cases of essential atrophy of the iris reported in the literature showed only holes or partial defect. A total disappearance of the iris, as far as we know, has not been reported before. We were fortunate enough to observe such a case recently.

Sun Chuan-Min, a man, aged 31 years, apparently had normal eyes when he was young, since neither his parents nor his friends ever made any remarks about them. His vision in both eyes was always good. Four years ago he suffered from a severe eye inflammation, which was probably trachoma. Since then his vision has been poor. Even three years ago he could read with the left eye alone.

On examination, both eyes were found to have trachoma, with scars in the conjunctiva of the upper tarsus and a thin total pannus. The right eye showed a normal anterior chamber, iris, and fundus. The left eye had a white macula on the temporal side of the cornea and had no iris at all. The depth of the anterior chamber could not be estimated. On the nasal side, the ciliary process could be seen projecting against the equator of the lens. On the temporal side, the corneal macula obstructed the view of the deeper structures. However, the golden glistening equator of the lens could be seen with focal illumination. The fundus was normal. The papilla was pink, the central vessels appeared normal, and there was no evidence of glaucoma. Vision was: R.E., 2/60; L.E., 1/60. The slitlamp examination was somewhat hindered on the temporal side by the corneal opacity and revealed a total absence of the left iris.

* From the Department of Ophthalmology of the West China Union University.

The visibility of the entire equator of the lens made the absence of the iris especially conspicuous.

We have to consider three possibilities for this absence: (1) Congenital aniridia. (2) Traumatic expulsion or extraction of the iris. (3) Essential atrophy.

As the patient's parents never mentioned anything about any anomaly of the left eye and as congenital aniridia would have been very conspicuous even in the darkly pigmented Chinese eye, since such an eye would have certainly shown a red pupillary reflex in the dark, it would seem that congenital aniridia was not present. That the patient had had good vision and could read with the left eye even as recently as three years ago further strengthens this contention. The second possibility, that of injury, is excluded by the negative history and by the clinical examination which did not reveal any sign of violence or scar.

Without doubt we have here a special case of essential atrophia iridis in which the disappearance of the iris tissue progressed so rapidly and so extensively that the last iridic fibers became invisible.

Essential atrophy of the iris is, as a rule, monocular as is exemplified in our case where the change was confined only to the left eye.

The tension of the right eye was 7 mm. Hg (Schiøtz) and that of the left eye, 16 mm. Hg. That the latter figure was much higher than the former again emphasizes the correctness of our supposition. The low tension in both eyes was probably due to the increased permeability of the cornea due to the pannus. Anyhow, the tension in the left eye was more than double that in the right.

If we take into account the probable permeable condition of the corneas, we may say that there was a definite relative glaucoma in the left eye. We know that in the later stages of essential atrophy the tension goes up. As a rule, it is at that time that a large part of the iris tissue is already resorbed. No doubt we have here in our case the cardinal sign necessary for the diagnosis of essential atrophy of the iris.

Concerning the cause of this increase in tension, there is much difference in opinion. We do not believe that the diminished surface of the iris due to the defects causes the resorption of the aqueous. We feel that the increase of tension may be due in early cases to the remnants of the fine fibers of the iris which are seen in some cases as white felt in the neighborhood of the holes of the iris or on the surface of the lens. These fibers may obliterate the gaps of the pectinate ligament and hinder the filtration in a way similar to that found in cases of glaucoma following degeneration of the zonular lamellae. Histologic examinations of cases with advanced glaucoma showed that the root of the iris was drawn to the pectinate ligament.[1]

Fine and Barkan's case[2] exhibited the most advanced degree of atrophy involving both eyes in a 9-year-old boy. The strands left formed a V-shaped figure in the right eye and an irregular X-shaped figure in the left eye. Duke-Elder[3] has summarized all the literature on this subject quite thoroughly. Rones reported a case where the iris showed an ectropion of the pigment epithelium; such an ectropion was apparently not present in our case.

E.E.N.T. Unit of the West China Union University Hospital.

References

1. Rones, B.: Essential atrophy of the iris. Am. J. Ophth., 23:163, 1940.
2. Fine, M., and Barkan, H.: Essential progressive iris atrophy. Am. J. Ophth., 20:277, 1937.
3. Duke-Elder, W. S.: Textbook of Ophthalmology. London, Henry Kimpton, 1945, v. 3, p. 2404.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLORADO OPHTHALMOLOGICAL SOCIETY

December 20, 1947

DR. MAURICE MARCOVE, *presiding*

FAMILIAL NODULAR DYSTROPHY

DR. WILLIAM M. BANE presented three cases of corneal dystrophy showing the deposits typical of Groenow's familial dystrophy including striations, small and large, round and oval deposits, ring-shaped opacities, and larger, white fluffy deposits. These cases were in the same family—the mother, Mrs. N., aged 39 years; a daughter, Betty, aged 14 years; and a son, John, aged 10 years. The youngest boy, aged 9 years, had normal vision and the corneas showed no signs of the disease. The mother stated that her mother and maternal grandmother, both deceased, had eyesight not improved much by glasses, and that she has two sisters living who have an eye condition similar to hers. Two brothers have no eye trouble. She and her family came from Iran. It seems safe to presume that the sisters and the mother and grandmother also had this same type of corneal dystrophy.

Mrs. N. states that, when examined 11 years ago in England, a diagnosis of punctate keratitis and vitreous opacities was made. The true condition was apparently not recognized. Betty and John had poor vision but never wore glasses and were never previously examined. Although examination with the corneal microscope revealed in each case a picture typical of the disease as described by previous observers, each of these three cases also showed a different picture. There were more striations in the 10-year-old child, the development of more nodules in the 14-year-old child, and more of the fluffy white deposits in the mother. It was also noted that, in the mother, there was more involvement of the deeper layers of the cornea than in the children. William Zentmayer in his article for the Jackson Birthday Volume, said that the disease was apparently limited to adults; whereas, Duke-Elder states that the earliest known cases begin about 10 years of age, although in one child aged five years, some early signs had been noted. The 10-year-old boy in this family had so much involvement of each cornea that it seems fair to believe that his eyes became involved when he was 8 or 9 years of age, or even younger.

Discussion. Dr. Harry W. Shankel quoted Berliner as saying that cases of corneal dystrophy are seen as early as five years. The cases herein presented are especially interesting in that they are seen in the typical stages of the various decades of life; in the youngest child, there are spots in radiating lines; in the older girl, these spots have coalesced somewhat; and in the mother they are seen in large groups of nodules. Dr. Marcove said that this is purely a degenerative disease and not at all infectious. It differs somewhat from Groenow's original report which was of an epithelial dystrophy.

CHORIORETINITIS DUE TO TOXOPLASMOSIS

DR. JAMES STRONG presented a white woman, aged 31 years, who had first been seen four months before complaining of blurring of right vision. She had had a similar attack four years before lasting five months and again three years before lasting three weeks. Examination revealed vision to be 20/25, O.U. The left eye seemed to be quite normal throughout. The right eye showed numerous keratic precipitates on the cornea, and the aqueous was quite full of cells. The iris, pupil and lens were normal.

The vitreous was hazy. An active area of chorioretinitis about 1 by 2.5 disc diameters in size was seen about 2 disc diameters above and temporal to the macula. Disc and vessels were normal.

The patient was started on atropine drops and penicillin by mouth, but the described signs became worse. Penicillin, 300,000u. in wax, was begun daily with no benefit. She was then hospitalized and given typhoid therapy alternating with calcium gluconate intravenously. She improved remarkably for a while and then became much worse again. She was given a course of gold sodium thiosulphate and improved considerably. In the meantime, a cataracta complicata had begun to develop. Vision was 20/200. One month ago she received a whole blood irradiation after which the lens cleared entirely, and the aqueous beam became almost imperceptible. X-ray films of the chest and an exhaustive physical examination were normal. A laboratory test for toxoplasmosis will be done soon.

Discussion: Dr. Ralph Danielson asked about the possibility of brucellosis and added that toxoplasmosis is a probability but not likely since it is seen mainly in children and only healed lesions are seen in adults. Dr. Walter Ohmart said that the negative X-ray films and Mantoux test did not finally rule out tuberculosis which this could well be. Dr. W. H. Crisp said that the probability of tuberculosis is so likely that, as a therapeutic measure, the patient should be at absolute bed rest for many weeks. Capt. A. Schultz suggested a therapeutic test of streptomycin, 1 gm. daily by injection or by iontophoresis, as it is given at Fitzsimons General Hospital with very good results. Dr. John C. Long stated that he was quite opposed to the use of gold empirically and quoted Hill of Richmond who reported the loss of both corneas after its use. Dr. Leonard Swigert repeated that the case seemed typically tuberculous and suggested a series of old tuberculin along with small doses of X ray with which he had had very good results in two cases of Eales's disease.

Dr. Strong (closing) agreed that all signs pointed toward tuberculosis but said that the internist on the case had insisted that infection could not possibly be present with all the performed tests being negative. Brucellosis tests had been negative. X-ray films of skull had shown reduction in brain convolutions which pointed toward toxoplasmosis. (Two weeks later, Dr. Strong received the report that this patient was strongly positive for toxoplasmosis.)

Bitemporal hemianopsia

Dr. James Strong presented a 23-year-old white woman who complained of headaches, blurring of vision, and eyestrain of six months' duration. In addition, she had had increased thirst for the past month. Examination revealed 20/20 vision, O.U., easily correctible to 20/15. Funduscopy was entirely normal except for blurring of disc margins in right eye. Visual-field studies revealed slight enlargement of each blindspot with moderate bitemporal hemianopsia. X-ray films of the skull were negative. Repeat of fields one month later showed that the hemianopsia had disappeared entirely. Impression: Optic neuritis.

Discussion. Dr. Marcove suggested that in optic neuritis of sufficient extent to blur disc margins, it would be most unusual to have 20/15 vision; therefore, the diagnosis is most unlikely and there must be some type of intrancranial pathologic condition. Dr. Long added that there should be a sedimentation rate and a spinal puncture to rule out an infection such as arachnoiditis.

Congenital glaucoma

Dr. Walter Ferer presented a 12-year-old Spanish boy who was admitted to Colorado General Hospital complaining of poor vision in the right eye during most of his life. The condition had been diagnosed as having hardening of the eyes but had received very little treatment. Examination

revealed vision to be: R.E., 20/400, incorrectible; L.E., 20/30, correctible to 20/20. Tension was 38 mm. Hg (Schiøtz) in the right eye and 34 mm. in the left. The corneas were large and prominent and measured 13 mm. The cornea of the left eye was clear, while that of the right was cloudy with many tears in Descemet's membrane. The irides were somewhat atrophic and the pupils were oval and reacted to light and accommodation. Anterior chambers and lenses were normal. The left disc was normal, while the right disc was deeply cupped and atrophic. Gonioscopy showed the right chamber angle to be entirely obliterated in all its circumference, while the left was obliterated only in patches. In the hospital, the tension of the left eye was easily controlled with pilocarpine and that of the right was not controlled at all with either pilocarpine or D.F.P.

Discussion. Dr. Marcove said that this case was presented for two reasons: (1) To seek surgical opinion as to type of operation to perform since something beyond medical treatment should be given, this would be a choice between goniotomy, trephination, and cyclodialysis; (2) to describe the drug D.F.P.

This drug was first discovered in a wargas plant where it was noted that those exposed to it became miotic and remained so for at least 10 days. At the University of Pennsylvania, it was found that the chemical was di-isopropyl fluorophosphate. It will soon be available commercially and is believed to be a tremendous advance in the medical treatment of glaucoma—especially secondary glaucoma. It was used at first in 0.1-percent solution but this seemed to produce too much spasm and 0.05-percent solution appeared to be much more practical. The drug stimulates the parasympathetics and its action is more prolonged than that of other miotics; however, it takes 2 to 3 days to commence its action after eserine or pilocarpine has been used. It is used in peanut oil and is unstable in water or mineral oil.

Dr. Danielson said that he had had a case almost identical with this one which was first seen when the patient was five months of age on which he had done a bilateral iridencleisis with very satisfactory results after three years. Dr. Tom Van Bergen said that at the University of California Clinics, the operation of choice in these cases is cyclodialysis.

Complication to streptomycin

Capt. A. Schultz (by invitation) said that since October, 1945, streptomycin therapy has been used at Fitzsimons General Hospital on slightly more than 500 patients with tuberculosis. In this group only one case with a visual-field defect was noted as a complication. This is a presentation of the case.

Mrs. M. L., a 31-year-old white woman, with pulmonary tuberculosis came to the Eye Clinic at Fitzsimons General Hospital, on October 20, 1947, complaining of sudden loss of vision in upper visual field of the right eye two days previously.

Her past history revealed that in July, 1946, a diagnosis of pulmonary tuberculosis was made. Her sputum was positive. The X-ray examination showed minimal infiltration of the right lower lobe. Cavitation was not found. She was treated with phrenic crush and pneumoperitoneum. The latter treatment consisted of the injection of about 800 cc. of air into the peritoneal cavity, five days before the onset of her visual symptoms. On August 28, 1947, parenteral streptomycin was begun, using 2 gm. daily in four divided doses. On October 18, 1947, 52 days after streptomycin had been started, the patient had sudden visual complaint, preceded by severe occipital headache. Streptomycin was discontinued on October 20, 1947.

Eye examination. On the first examination on October 20, 1947, visual acuity in the right eye was 20/30, correctible to 20/20; in the left eye, 20/20. The pupils were equal and regular, reacted well to light and accommodation. There was no diplopia. Slitlamp

examination was negative. Ophthalmoscopic examination of the right eye revealed the media to be clear, disc normal, retinal arteries generally attenuated, superior vessels slightly, inferior vessels moderately. The macula was normal, as was the periphery. The patient could not see the ophthalmoscopic beam in the inferior retina. The left eye was normal throughout. There was no caliber irregularity of arteries.

Visual field. Right eye, absolute altitudinal defect for 3/300 white and 3/1000 white up to a bright light through midline, sparing macula. There was a relative area in entire lower half of field for 3/330 and 3/1,000 white.

Color sense. She could recognize 10/1,000 blue within 10 degrees of macula inferiorly in the right eye, but no red or green. The left eye was normal.

Therapy. Immediate therapy consisted of 50 mgm. sodium nitrite, I.V., (Nitroscleran) and 50 mgm. q3h, I.M., for 24 hours. Hourly massage of the eye was ordered. The following day it was felt more energetic therapy was indicated since the vascular spasm and field defect persisted. The ciliary ganglion was anesthetized with 2 cc. of 2-percent novocaine injected retrobulbarly. A paracentesis of the anterior chamber was performed. An injection of 100 mgm. sodium nitrite, I.V., was made concurrently. Dicumarol was started, keeping the prothrombin time at 40 to 50 percent of normal; 660 mgm. rutin were given daily. The latter two drugs were given on the premise that a thrombus might be present at the bifurcation of the central retinal artery.

Course. Under this regimen, the field defect improved and recognition of colors returned. On October 23, 1947, three days after onset, ophthalmoscopic examination of the right eye disclosed about one diopter of papilledema with enlargement of the blindspot. Visual acuity remained at 20/30 in the right eye, correctible to 20/20 until November 7, 1947, 19 days later, when visual acuity in the right eye was found to be 20/100, uncorrectible. No new ophthalmoscopic findings were present. Previous therapy was continued. Four ampules of amyl nitrite were inhaled, improving vision to 20/70 in the right eye. This was repeated on alternate days and in two weeks visual acuity in the right eye was 20/30 uncorrected. The final examination on December 2, 1947, showed visual acuity of the right eye to be 20/30, correctible to 20/25; left eye, 20/20.

Ophthalmoscopic examination of right eye showed the media to be clear; disc was of normal size, shape, and color except for a slight temporal pallor; the previous elevation had disappeared. The arteries of superior retina were nearly normal in caliber, A-V ratio was 2:3. The inferior retinal arteries showed generalized attenuation but were not as constricted as at the time of first examination. A-V ratio, 1:3. Macula and peripheral fundus normal. Left eye entirely normal.

Visual field. Visual-field studies showed the right eye to have a normal peripheral field for 3/330 white. Central field (3/1,000 white) showed normal physiologic scotoma, but a 2 by 5 mm. vertical relative scotoma was present just nasal to the fixation point. This was a remnant of the previous superior scotoma. Color fields for 10/1,000 red, green, and blue, were almost normal.

The patient has had no recurrence to date. Drug therapy was discontinued on November 20, 1947. No more streptomycin has been given. Pulmonary tuberculosis improved under the relative short course of streptomycin therapy.

Discussion. Dr. Long said that intravenous ether has been used in arterial emboli in legs and it might have been useful here. Dr. Marcove said that he felt that the ganglion cells of retina die after 3 to 4 hours. It was unusual that in this case amyl nitrite benefited after 17 days.

Morris Kaplan,
Secretary.

SOCIEDAD OFTALMOLOGICA DE MADRID

June 26, 1947

Entropion of the lower lid

Dr. Marin Amat presented his operative procedure, consisting of: (1) An incision parallel to the ciliary border and some 5 mm. from it, including the skin and the orbicularis muscle; (2) resection of the muscle fibers if they protrude in the wound; and (3) placement of the suture points (which is the fundamental part in the procedure) so that the needle point penetrates successively the lower (cutaneous) lip of the wound, deep enough to include the thickness of the broad ligament, and the upper (cutaneous) lip of the same wound. In addition some auxiliary steps are taken when necessary.

This method, in addition to its simplicity and rapidity of execution, has the great advantage in that the sutures can be removed a day or two after the operation, giving a perfect functional and cosmetic cure.

Discussion. Dr. Mario Esteban said that in order to have an operation for entropion give desirable results it is always necessary to look for a base to give firm support to the sutures, a matter which is as easy in the upper lid as it is difficult in the lower lid. In the lower lid where the tarsus is narrow and flexible we can only find tarsal support if we make the incision and the line of suture very near to the free border. Sometimes the best way to achieve this is simply to make a deep lineal cauterization parallel to the ciliary border.

The usual operations with or without excision of a cuneiform strip or an elliptical piece, and with sutures placed beneath in superficial planes, are of uncertain results in the lower lid. Sometimes the skin will slip upwards and nothing will have been accomplished. Other times there will be a shortening and descent of the lid, but no eversion of its border and the eyelashes.

I believe that the procedure described by Dr. Marin Amat obviates the first of these inconveniences. As for the second, one must be careful to give the suture threads an adequate direction so that when tying, the palpebral border will be drawn forward and not downward.

Dr. Marin Amat concluded by thanking Dr. Esteban for his discussion. He added that this procedure, which he had used many times, does not give safe results and that the cure is obtained much later after elimination of the scab and cicatrization by second intention. The effect of the suture is so great that one must estimate it carefully so as not to produce an ectropion.

Pseudoneoplasms of the orbit

Dr. Diaz Gomes and Dr. Marin Amat discussed two cases of this condition, one of a chronic inflammatory type and the other of a hydatid cyst.

The first case is that of a 48-year-old woman who showed an enormous exophthalmos (10 mm.) of the right eye which was not reducible, did not pulsate, and had developed slowly. All biologic tests (Wassermann, the reactions of Weinberg and of Cassoni) were negative, as well as X-ray studies. A very extensive Krönlein operation showed an extensive flat neoplasm in the orbit, situated between the bony and muscular pyramid. It completely covered the latter on the outside, above and within.

The cure was rapid with proper position of the globe in the orbit, return of vision, and disappearance of the intense papillitis that had existed. There only remained a slight immobility of the globe and ptosis of the upper lid. Histopathologic analysis showed that it was a case of chronic inflammatory nature of the acinous glandular type (lacrimal gland) with infiltration of lymphocytes, plasma cells, and histoid elements.

The second case was that of a boy, aged 16 years, who had an exophthalmos (6 mm.) of the left eye for several months, irreducible and without pulsation, with almost complete loss of sight and a very intense papillitis. It was thought this might

be an intraorbital neoplasm, perhaps of the optic nerve, in view of the age of the patient and the early loss of vision.

In order to expose the neoplasm freely, a transfrontal surgical procedure was undertaken, which confirmed the presence, at the vertex of the orbit above and within the optic foramen, of a hydatid cyst the size of a chestnut. This was perfectly and fully removed. The postoperative course was satisfactory. The patient left the hospital without exophthalmos, without diplopia, with normal ocular motility, with a vision of 6/6, and with complete disappearance of the papillitis. There was a perfect anatomic and functional cure.

The extensive surgical procedures permitted to remove completely the neoplasms, in the first despite its extension and in the second despite its depth, are factors which are essential in operations of the orbit. Dr. Diaz Gomez made a few additional observations. In the first place it is very useful in order to diagnose the exact localization of the tumor and therefore to determine the best way to approach it, to get exact visualization of all the structures by means of injecting in the orbit some opaque medium of contrast to the X rays, a medium which would be harmless to the eye and the other intraorbital structures. He added, "I have read somewhere about this procedure, but do not remember where nor the details and I will be grateful to any of you gentlemen who can enlighten me on this."

The second point is that the transfrontal route could even be improved, in the sense of avoiding loss of the superior orbital border with disagreeable cosmetic consequences, as well as the difficulties which the resection places on the muscles which are inserted there. I believe this could be avoided by omitting to resect a strip of the orbital border, and, if this is not possible, it might be possible to insert a strip of bone afterwards to replace the lost bone, similar to the other procedures we do in cranioplasty.

Discussion. Dr. Bartolozzi congratulated Dr. Marin Amat and Dr. Diaz Gomez for their very interesting paper. The orbit is a closed box and to diagnose what takes place there is one of the most difficult things in ophthalmology. For this reason many times the only solution is to open up the box in the manner which Dr. Diaz Gomez has done with such brilliant results. It is evident that it is difficult to diagnose correctly many cases of exophthalmos especially when one lacks sufficient experience.

I recall a case of exophthalmos seen with Professor Carreras and diagnosed by him as having a chronic inflammatory origin. In this case, there was no antecedent factor except an old trauma to the frontal region. Could this be one of those rare cases where the process was due to a late hemorrhage caused by changes due to a very old trauma? At times, several years may elapse between the trauma and the hemorrhage. Only a surgical intervention could have cleared this up.

With reference to the suggestion of Dr. Diaz Gomez on the possibility of obtaining radiographs of the orbits with their contents visualized by means of a contrast medium, this work has already been realized. Finally, I agree with Dr. Diaz Gomez on the desirability of trying a plastic reconstruction of the roof of the orbit in cases where it is necessary to remove it in the course of the operation, since this would improve the appearance of this region and the function of the musculature.

Dr. Marin Amat thanked Dr. Bartolozzi for his discussion and added that the Krönlein operation which Dr. Diaz Gomez performed on the first patient is not the limited, circumscribed operation which we oculists do, but it is a much more extensive operation, with removal of the entire external wall of the orbit and, in addition, a large part of the roof of the orbit down to the sphenoidal fissure, so that the full contents of the orbit can be explored and the extensive tumor can be removed. In his second case, the transfrontal approach, he mobilized a

very extensive piece of bone periosteum and muscle, leaving it to adhere by its lower-external portion. Resection of the whole roof of the orbit leading to the exposure of the frontal lobe of the cerebrum and all orbital contents affords a large field of observation by means of which it is not only possible to remove the hydatid cyst from the apex of the orbit but also to perform total extirpation in cases where it invades the cranial cavity.

Facial paralysis with hysterical manifestations

Dr. Bartolozzi presented a clinical case characterized by the association of facial paralysis with typical hysterical manifestations. It is a case of a young woman, aged 18 years, who suddenly showed the following symptoms: deviation of the mouth towards the right side, impossibility of closing the left eye, pain in the left half of the head, and marked loss of vision of the left eye. The first examination showed a left, total facial paralysis, anesthesia of the regions supplied by the ophthalmic and superior maxillary branches of the trigeminus, limitation of movement of the left eye (downward, downward and inward, and downward and outward), marked loss of vision of this eye (fingers at half a meter) and slight loss in the right eye (7/10). The visual fields were restricted in both eyes, more marked in the left. The fundus was completely normal. Other tests, ocular and general, were negative. Not being able to ascribe such a complex picture to some definite process, it was suspected that the case presented a superposition of an hysterical process upon an organic picture. When the test was properly made, a typical "fatigue" field was obtained. Concurrently there was a rapid improvement in visual acuity.

Discussion. Dr. Marin Amat congratulated Dr. Bartolozzi and confirmed the extreme symptoms outlined, mentioning the frequency with which hysterical manifestations are superposed on organic diseases. This frequency is even more manifest in diseases of the nervous system, necessitating a careful discrimination between the functional or hysterical symptoms and those due to an organic or anatomic lesion.

When the second pair of cranial nerves (optic nerves) are affected at the same time with another pair of cranial nerves, as in the case of Dr. Bartolozzi, the functional examination will remove all doubts and will disclose the hysterical manifestations superposed on an organic lesion. In cases where vision is retained, the problem is simple, assuming that the hysteria shows the triad described by Charcot—a spreading of the personality, reduced field of consciousness, and a reduced field of vision.

One should emphasize that the constriction of the field of vision is one of the most constant phenomena of ocular hysterical manifestations. The constriction is concentric and regular and has certain characteristics which help to reveal it. These are: an inversion of the red with reference to white; the displacement curves of Foerster, and the fatigue curves of Wilbrand, or the fatigue spirals of Von Reuss, the outlines and isopters of which constitute true spirals like those which Dr. Bartolozzi has shown in his patient. In addition, the concentric reduction of the visual field shows a constant variability not only from day to day but from one moment to another. These are typical changes in the visual fields of hysterical and neurasthenic patients.

On the other hand when there is loss of visual acuity (amaurosis) the diagnosis is also not difficult (pupillary reflexes and fundus being normal). However, there are exceptional cases where there could coexist an amaurosis due to an organic lesion in the cortical centers of sight (as uremia or lead poisoning, principally) when it becomes necessary to resort to the other symptoms of these affections in order to complete the diagnosis.

Joseph I. Pascal,
Translator.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

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THE REBATE CASES

By this time everyone knows that 3,221 ophthalmologists in the United States have been charged in the antitrust suits brought by the government against the Bausch and Lomb Optical Company and four subsidiaries and the American Optical Company with violating the Sherman antitrust law by accepting rebates. That the number is so large should occasion little surprise, for it has long been known that the practice of accepting rebates by ophthalmologists is extensive, hoary with age, and has almost become a tradition or at least an accepted custom.

This custom arose in the very early days of American medicine when almost every physician and surgeon accepted rebates from drug stores, undertakers, and orthopedic supply firms, as well as from opticians. Fee splitting between physicians and surgeons was common and widespread. Specious arguments in favor of such practices have been advanced from time to time among the doctors themselves. That they were uncomfortable about it, to say the least, is shown

by the fact that their patients knew little or nothing about what went on behind the scenes.

Physicians of every specialty, furthermore, took hypocritical pleasure in condemning openly and officially in their societies and special organizations these usages as unethical. As has been said before, it was like Congress and the people during Prohibition days, voting dry but drinking wet. We look back on those evil days of national hypocrisy with no pleasure to our conscience.

Fee splitting still goes on, but certainly in a very subdued and gradually disintegrating form. The optical rebate practice is the last to remain of these unethical ways of getting something for nothing by exploitation of the patient. Thanks to government action it, too, appears to be on the way out. Unfortunately, the great science of ophthalmology and all ophthalmologists in this country are smeared and stained with muck in the process. The price will be worth it if the cancer is excised.

Among the 3,221 ophthalmologists named in the suit are members of all the ophthalmological organizations of this country, including the American Board of Ophthalmology. These are our friends and respected colleagues. Many of them are our leaders whose contributions to our science are noteworthy and significant, whose service to their patients is of the highest quality, and whose personal conduct is above reproach. We know that they and the rest of us deep in our hearts have hated the rebate practice that has so many roots in our professional lives and that has become so firmly established through effective outside pressure and reluctant inner complacence.

A widespread custom, handed down from generation to generation of ophthalmologists, endorsed and engaged in by so many of our past leaders and present prominent figures, overwhelmingly seductive to our young men just getting started in practice and eagerly fostered by optical firms, cannot be expected to be easily changed, especially by courageous, individual voluntary action.

It is significant, therefore, that 92 members of the Chicago Ophthalmological Society named in the antitrust suit have accepted a consent decree recently entered in the Federal District Court at Chicago. Under the terms of the decree, the ophthalmologists, while not admitting receipt of rebates from the optical companies on the sale of spectacles to their patients, consent to be enjoined against accepting such rebates and to remove themselves from any further proceedings in the antitrust suit against the optical companies.

The stipulation stated that the physicians are in agreement in condemning rebates by optical companies, that they affirm that they are not accepting such rebates, and pledge themselves not to accept any rebates or credits from dispensers. The order that was issued also provides that other doctors named in the suit may voluntarily become parties to the consent decree by signing the stipulation.

The action taken by the members of the Chicago Ophthalmological Society is a welcome and wholesome sign of moral progress. It means that they will no longer be glorified optometrists, but true doctors of medicine. It means that they will practice ophthalmology unfettered by commercial chains. It means considerable financial loss to their pockets, but it means increasing confidence, love, and respect from their patients, and quiet nights of respect for themselves.

Derrick Vail

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjuctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Spring, K. H., and Stiles, W. S. **Apparent shape and size of the pupil viewed obliquely.** Brit. J. Ophth. **32**:347-354, June, 1948.

After photographing pupils at varying angles the authors found considerable individual variation in the apparent position of the pupil at oblique angles of view. For all pupil sizes the relative apparent vertical diameters at different angles are the same. At a given angle the ratio of horizontal to vertical diameters may be taken as a linear function of the apparent pupillary diameter for normal view.

Orwyn H. Ellis.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Braley, A. S. **Experimental studies in inclusion blennorrhea. Isolation of a virus.** Tr. Am. Ophth. Soc. **45**:432-461, 1947.

It is assumed that a new unknown virus was isolated, which however may represent a variant of the virus of inclusion blennorrhea. Because of the manner in which the inclusion blennorrhea was handled in passing through the mouse brain it is quite possible that a mutation of the virus may have occurred, which transformed an atypical virus into a typical one. This virus could then give rise to a follicular conjunctivitis in baboons in which there might be an immunity to further innoculation with the inclusion blennorrhea from a patient. If we consider a virus to be a mutation from bacteria by loss of their ability to synthesize artificial media then it would appear probable that a mutation of an atypical virus could be brought about.

It is concluded that the virus of inclusion conjunctivitis as it occurs in the human was not grown, but a variant which was a true virus was produced from the atypical virus of inclusion blennorrhea.

David O. Harrington.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Estable, J. J. **The ocular effect of several irritant drugs applied directly to the conjunctiva.** Am. J. Ophth. **31**:837-844, July, 1948. (2 figures, 14 references.)

Palm, Eric. **On the passage of ethyl alcohol from the blood into the aqueous**

humour. Acta ophth. **25**:139-164, 1947.

The purpose of this work was to investigate how a lipoid-soluble substance would pass into the eye. Ethyl alcohol was chosen as the typical substance. It was injected into animals, the changes in the alcohol concentration in the blood and the aqueous humour were measured at intervals and the two curves thus obtained were measured. Alcohol passes through the membrane that separates the blood from the aqueous at so high a rate that diffusion in the anterior chamber cannot be regarded as taking place immediately and consequently the rate of penetration cannot be determined precisely. The penetration rate is estimated to be approximately one-third to one-half of the rate at which heavy water enters the eye. Alcohol enters the eye more rapidly than any other substance than water; for example at five to eight times the rate of urea. The comparison was suggested that the membrane that separates the blood from the eye functions, as far as permeability is concerned, as a porous lipoid membrane. The high rate of penetration of alcohol as compared with that of previously investigated lipoid-insoluble substances shows that the conditions that prevail in the eye are similar in this respect to those in the central nervous system and differ fundamentally from those found in the other tissues of the body.

F. H. Haessler.

Pirie, A., Schmidt, G., and Waters, J. W. **Ox vitreous humour. 1. The residual protein.** Brit. J. Ophth. **32**:321-339, June, 1948.

The authors review the scanty literature on vitreous residual protein, and present their methods of procedure. Partition chromatographs of hydrolysates of commercial gelatin, cornea gelatin, vitreous humour residual protein and a mixture of glycine, proline and hydroxyproline showed striking similarities in the pattern exhibited by the first three. It was evident that all three contained roughly comparable amounts of proline, hydroxyproline, glycine, glutamic and aspartic acids, and alanine. This evidence was sufficient to suggest that the vitreous humour protein might be a member of the collagen gelatin group. X-ray photographs of vitreous humour residues, both in the unstretched and the stretched state were taken. The general agreement of the spacings of the X-ray reflections with those of oriented collagen fibers provides additional support for the view that the protein of the vitreous humour belongs to the collagen class. For further identification the action of collagenase which digests only collagen and reticulin of proteins was studied. A preparation of the enzyme readily dissolved the isolated residual protein of the vitreous humour. This again suggested that the protein was of the collagen class. The structure of the vitreous humour, which may perhaps be considered as a network of submicroscopic fibers, has a firmer, more stable arrangement than a colloidal gel.

When comparing the swelling curve of ox cornea collagen with the vitreous as a whole, acidity or alkalinity was found to have the exactly opposite effect. The effect of salts on the volume of washed vitreous humour residues was studied. The salts were found to depress shrinkage, and in general the valency effect of the anion was more clearly apparent. Ox vitreous humour has been shown to have a great deal more hexosamine than the humours of other animals. This indicates that the hyaluronic acid content of ox vitreous humour is greater and this probably explains its greater rigidity.

Vitreous cannot be considered as uniform, but must be made up of at least two structures, the collagen-like network and the hyaluronic acid and protein jelly. Liquefaction of the humour follows enzymic hydrolysis of the network, and therefore one may say that this network is essential for maintenance of a normal

vitreous humour. The relationship of the network to the jelly is of prime importance. Orwyn H. Ellis.

Spring, K. H., and Stiles, W. S. **Variation of pupil size with change in the angle at which the light stimulus strikes the retina.** Brit. J. Ophth. **32**:344, June, 1948.

No abnormally high pupillomotor effect resulted from rays that entered near the edge of the dilated pupil, and hence were incident on the retina at an angle with the normal. Thus, for practical purposes the part of the pupil through which light enters the eye is not a factor in determining size of the pupil. Orwyn H. Ellis.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Freusberg, O. **Muscular spasm of accommodation or transitory lenticular myopia in a swampfever-like toxemia.** Klin. Monatsbl. f. Augenh. **112**:304-308, 1947.

The clinical syndrome of high fever, nausea, marked albuminuria and transitory myopia was observed among German troops stationed in the swamps of northern Finland. The author denies the possibility that this myopia could be due to a central or peripheral toxic stimulation of the accommodative mechanism, because the myopia does not disappear under the influence of a mydriatic and because of the lack of a simultaneous miosis. He regards the myopia as a lenticular one due to changes in the refractive index in the lens, similar to the transitory myopia in diabetes and in sulfonamide poisoning. (References.) Max Hirschfelder.

Harms, H. **The distribution of the light sense in the retina.** Klin. Monatsbl. f. Augenh. **112**:353-357, 1947.

The light sensitivity of various meridians of the retina during light and dark adaptation was investigated by the author. In light adapted persons the fovea is found to have the highest sensitivity, whereas the peripheral parts show ten to fifteen times lesser sensitivity. The greatest decrease takes place within a radius of 20 degrees from the center of the retina. The fovea centralis of dark adapted persons shows less light sensitivity than its immediately adjoining region, usually 5 to 15 degrees from the point of fixation. The relative increase of light sensitivity in dark-adapted persons is more marked within this intermediate zone than it is in more peripheral parts. Individual differences in the various persons tested were noted during the experiments. (References.) Max Hirschfelder.

Jaeger, A. **A new instrument for checking the visual acuity in reduced illumination.** Klin. Montsbl. f. Augenh. **112**:346-353, 1947.

A "nightvision selector (Nachtsehwähler)" is described which has the advantage of greater flexibility during the examination of acuity in low grade illumination. The instrument permits a change in the intensity of the illumination as well as a change in the size of the test letters. The article describes the use of a luminous foil for gauging the intensity of illumination, the arrangement of the illuminating lamps, and the test charts used. It also contains the arrangement which provides for constant light adaptation which precedes the test for vision in reduced illumination. In practice the test is taken by several persons at once, who sit 5 m. from the test object and are exposed to illuminated test signs of 0.005 lux and 0.0005 lux intensity for 10 seconds after various time intervals. After 15-minute dark adaptation and with 0.0005 intensity 61 percent saw about 0.09, 30 percent 0.06 and 8 percent less than 0.05. (References.) Max Hirschfelder.

Katz, J. **A new method for exact determination of visual acuity.** Klin. Monatsbl. f. Augenh. **112**:341-346, 1947.

A method for exact determination of visual acuity by means of rotable Landolt rings is described. The method excludes the influence of guessing by making mathematical allowance for results which are subject to guess. Twelve observations of each of two to four Landolt rings of various sizes are the basis of the method. The article contains the mathematical calculations which make the method an exact one. Max Hirschfelder.

Kessler, J. **Instrument for the measurement of depth perception.** Klin. Monatsbl. f. Augenh. **112**:339-341, 1947.

Two discs of glass, one of which is transparent, the other translucent, are arranged so that the distance between them can be varied. The transparent glass bears points arranged to make a letter or number, the other has an irregular arrangement of dots. A source of light is placed behind them. The measure of depth perception is the smallest angle of separation under which the patient can recognize the figures on the anterior disc.

Max Hirschfelder.

Swan, K. C. **Some aspects of studying binocular vision.** Am. J. Ophth. **31**:845-852, July, 1948. (3 figures, 5 references.)

v. Tschermak-Seysenegg, A. **Is Fraunhofer correct?** Klin. Monatsbl. f. Augenh. **112**:358-360, 1947.

The arrangement of slit and prism for the dispersion of light into spectral colors puts red to the left, blue to the right side. The letters A to H signifying the Fraunhofer lines are arranged in the same manner. England and America follow the opposite orientation and arrange the spectroscope so that blue is on the left, red on the right. According to the author, this is done to have the lower wave length on the left, the higher one on the right and, in this way, have an increasing mathematical series instead of a decreasing one. The author proposes a compromise which would retain the original arrangement by Fraunhofer which is still in use in continental Europe, but which would introduce the usage of the Hertz oscillation numbers which are low for red and high for blue.

Max Hirschfelder.

5

DIAGNOSIS AND THERAPY

Heinz, K. **The totally reflecting prism as an adjuvant in ophthalmoscopy.** Proc. Ophth. Soc. Vienna p. 90, Dec. 11, 1944.

By the use of a rectangular prism with reflecting hypotenuse surface it is possible to examine even the most distant periphery of the fundus of the human eye. Neither the patient nor the examining physician have to change their mutual positions during the examination. It is sufficient to turn the prism in different angles in order to visualize the distant sections of the periphery of the fundus. The instrument is of value in localizing retinal holes. F. Nelson.

Kirby, D. B. **Instruments for cataract surgery.** Tr. Am. Ophth. Soc. **45**:423-432, 1947.

Detailed specifications for a complete set of matched instruments for cataract surgery is presented. All the instruments have balanced cylindrical handles so that each instrument has the same feel, grip, length, weight, size and balance. The article is photographically illustrated.

David O. Harrington.

Koch, F. L. P. **Pupillometry: a comment.** Tr. Am. Ophth. Soc. **45**:415-422, 1947.

The various techniques of pupillometry and pupilography are reviewed in considerable detail. The characteristic components of the normal light reflex are listed and the application of pupillometry to diagnosis is discussed.

David O. Harrington.

Moehle, Walter. **Corneal scissors.** Am. J. Ophth. **31**:864-865, July, 1948. (1 figure.)

Sloan, L. L. **A note on plastic test objects for perimetry.** Am. J. Ophth. **31**:861-862, July, 1948. (1 table.)

6

OCULAR MOTILITY

Coachman, E. H. **A common extraocular muscle imbalance.** Eye, Ear, Nose and Throat Monthly. **27**:175-179, April, 1948.

The author emphasizes the necessity for prolonged occlusion to detect the total muscle imbalance because momentary screening will not bring out latent phorias. In the vertical phorias, especially, this is essential for complete correction and relief of symptoms.

Orwyn H. Ellis.

Critchley, C. F., and Cameron, A. J. **Exophthalmic ophthalmoplegia treated with prostigmin.** Lancet **1**:751, May 15, 1948.

A 33-year-old woman with exophthalmic ophthalmoplegia was treated with daily injections of 0.125 g. of prostigmin for two months. The exophthalmos and ophthalmoplegia improved and the dose of prostigmin was reduced. In two weeks one eye became more protuberant, 0.125 g. was again given, and the proptosis subsided. After about seven months the prostigmin was withheld. A month later the proptosis appeared to have decreased a little more and the function of the eye was otherwise within normal limits. (References.) Bennett W. Muir.

Jackson, F. E. **Problems in research in orthoptics.** Am. J. Ophth. **31**:805-810, July, 1948. (2 figures, 1 table.)

Jaensch, P. A. **Ocular muscle implants.** Klin. Montsbl. f. Augenh. **112**:309-312, 1947.

This article is a supplement to a previously (vol. 111) published paper on the same subject. The results of the Hummelsheim operation in 12 cases of congenital or acquired paralysis of the lateral and medial rectus are described. The author recommends the procedure for all cases of paralysis of the lateral muscle. The usefulness in cases of paralysis of the medial muscle needs further investigation. In cases of postoperative divergent squint, however, a muscle implant from the upper and lower rectus muscle supports and supplements the advancement of the internal rectus. (References.)

Max Hirschfelder.

Scobee, R. G. **Anatomic factors in the etiology of heterotropia.** Am. J. Ophth. **31**:781-795, July, 1948. (11 figures, 6 references.)

Schulte, D. **Bilateral isolated paresis of the superior rectus, a rare disturbance of the eye muscles.** Klin. Monatsbl. f. Augenh. **112**:299-304, 1947.

The author observed a bilateral isolated paresis of the superior rectus muscle. Upward rotation and torsion were disturbed in the position of abduction of the individual muscle. The resulting double images are like those due to bilateral overaction of the inferior oblique muscle. The cause of the paresis was probably vascular damage in both lateral nuclei of the oculomotor nerve. The most sensible treatment of the condition is bilateral tenotomy of the inferior oblique rather than a bilateral recession of the inferior rectus muscle. Max Hirschfelder.

Venco, Luigi. **On the factors which regulate the taking of corneal transplants.** Ann. di ottal. e clin. ocul. **72**:749-766, Dec., 1946; **73**:28-52, Jan., 1947.

Venco reviews the pertinent literature and discusses at length the various factors which may possibly bear on the taking of corneal transplants. He holds that success in keratoplasty is due above all to the great vitality and resistance of corneal

tissue, to its relatively low rate of metabolism, to its marked regenerative capacity, and to the fact that the transplant is in immediate contact with its sources of nutrition.

There is evidence that the cornea has greater resistance than any other tissue known to biologists and that, because of its low rate of metabolism, it can survive for some time even after refrigeration and when deprived of its external sources of nourishment. Likewise the grafted tissue is well tolerated because the corneal proteins (presumably because of a deficiency of closed-chain amino acids in their chemical composition) have little or no antigenic property.

The orientation or "polarity" of the transplant may be entirely disregarded. Corresponding layers in host and transplant must be apposed as accurately as possible, but the graft may be placed without regard to its original meridians in the donor's eye. The presence of normal tissue in some part of the surrounding cornea of the recipient's eye is essential if the graft is to remain transparent. Whether the donor and recipient are related by blood or whether they belong to the same blood group is immaterial. Since in general the factors that might prevent a successful transplant diminish with age, the older the donor the greater the chances of success.

Harry K. Messenger.

7

CONJUNCTIVA, CORNEA, SCLERA

Brausewetter, G. **A case of band-shaped corneal opacity successfully treated with hydrochloric acid.** Proc. Ophth. Soc. Vienna p. 44, March 29, 1943.

A posttraumatic band-shaped extensive corneal opacity in a patient's eye was treated twice with 2-percent hydrochloric acid solution after removal of the epithelium. The vision improved from counting of fingers at 25 cm. to 6/36.

F. Nelson.

Bruens, E. **Treatment of gonorrheal ophthalmia with chemotherapeutic and antibiotic remedies.** Klin. Montsbl. f. Augenh. 112:327-332, 1947.

The sulfonamide therapy of the past eight years is reviewed. It is pointed out that the successful treatment of ophthalmia neonatorum by the exclusive use of sulfonamides during the earlier years has become less and less successful lately, because the gonococci became sulfa-resistant or because only sulfa-resistant strains survived the influx of the new drug. There are indications that the treatment with penicillin will suffer a similar experience. The author recommends quarter-hourly irrigation of the conjunctival sac with normal saline solution in addition to the half-hourly instillation of 20 percent Cibazol or Globucid (sulfathiazole compounds) solution or repeated instillation of penicillin solution. For severe cases he suggests general sulfa treatment and intramuscular milk injections, again in addition to the regular quarter-hourly irrigation of the conjunctival sac. (References.)

Max Hirschfelder.

Giacomelli, Piero. **The rosaceal keratidites.** Arch. di ottal. 51:106-135, July-Aug.-Sept., 1947.

The author describes two cases of keratitis rosacea, briefly summarizes the clinical picture, and reviews the principal hypotheses advanced to explain this obscure problem. The disease is an expression of a decreased respiratory activity in the tissue which is brought on by an increase in venous pressure and by deficiency of lactoflavin. This is probably due to a disfuction of the suprarenal cortex which controls both these factors.

Francis P. Guida.

Hatschek, G. **Contribution to the treatment of diseases of the anterior segment.** Klin. Monatsbl. f. Augenh. 112:318-327, 1947.

The therapeutic measures for various

forms of conjunctivitis, blepharitis, corneal ulcers and herpes corneae are discussed. The lack of certain drugs in postwar Europe has been taken into account. The measures which are recommended follow in general those used in the United States. The use of silver nitrate for conjunctivitis is strongly stressed. Subconjunctival injection of the patient's blood (3 to 5 cc.), puncture of the anterior chamber and parenteral injection of the hormone of the adrenal glands has been of value in the treatment of cases of deep keratitis which resisted ordinary measures. The importance of improving the general health of the patient by adequate general measures is stressed. (References.) Max Hirschfelder.

Lepri, Giuseppi. **Epithelial sclerocorneal cysts of congenital origin associated with congenital malformation of the fundus.** Arch. di ottal. **51**:53-80, April-May-June, 1947.

Lepri reviews the literature and then describes the general characteristics and accepted pathogeneses of non-traumatic sclerocorneal cysts. He describes one case of congenital cyst which showed an associated prepapillary cyst in the left eye and a very small cyst on the nerve margin of the right eye. Francis P. Guida.

Lindner, K. **Excision of corneal scars.** Proc. Ophth. Soc. Vienna, p. 72, May 22, 1944.

A woman, 40 years of age, had frequent severe eye inflammations during childhood resulting in dense central keloid scars in each cornea. Vision was reduced to counting of fingers at 25 cm. After superficial trephining with a 3-mm. trephine the keloids, together with an opaque layer of corneal tissue, were "pealed off." In the right eye a vision of 6/36 was obtained with contact lens. F. Nelson.

Lindner, K. **The treatment of a case of argyrosis of the conjunctiva.** Proc. Ophth. Soc. Vienna p. 69, April 17, 1944.

Following the procedure of Weymann (J.A.M.A. 1367, 1929) Lindner treated an opera singer who had developed a disfigured argyrosis of the conjunctiva with subconjunctival injections of a mixture of 12-percent sodium thiosulfate and 2-percent potassium ferric cyanide. It is necessary to use platinum-iridium needles for these injections. Four treatments were necessary and the patient was fully satisfied with the cosmetic result.

F. Nelson.

Lippi, E. V. **White rings of Coats.** Arch. di ottal. **51**:97-105, July-Aug.-Sept., 1947.

The author describes one case and then presents his theory of the pathogenesis of the lesion. He believes that there is an alteration of the perforating nerves of the cornea and of the openings in Bowman's membrane which leads to a degenerative process of the surrounding tissue. This theory of pathogenesis does not preclude a neurovascular etiology. To a primary alteration of this nature a toxic action or a trophic disturbance may be added.

Francis P. Guida.

Michtner, H. **Uncommon lead impregnation of the cornea.** Proc. Ophth. Soc. Vienna p. 87, Dec. 11, 1944.

An 18-year-old woman who had been in an explosion of lead nitroresorcinate suffered burns of the upper extremities and the thorax as well as foreign body lesions in both eyes. The bulbar conjunctiva and the lower temporal quadrant of the cornea of the right eye had numerous small grayish-white opacities in the epithelial layer. Vision was 6/6. The left eye also had numerous opacities in the exposed section of the conjunctiva. The cornea was dotted with numerous dust-like grayish-white incrustations which were confluent in places and also involved the parenchyma. The structure of the iris was blurred and somewhat hyperemic. The incrustations probably were lead oxide. Microanalysis of particles of the corneal

opacities by a spectrographic method failed to show lead. The author tried to dissolve the opacities which, in later stages, probably consist of lead carbonate, by application of ammonium tartrate.

F. Nelson.

Pendexter, S. E., Jr. **B. pyocyaneus corneal ulcer treated with penicillin.** Am. J. Ophth. **31**:862-863, July, 1948.

Rössler, F. **Psoriasis of the conjunctiva.** Proc. Ophth. Soc. Vienna p. 42, Feb. 22, 1943.

A woman, 29 years of age, had generalized psoriasis on the face, forehead and upper and lower lids as well as on the trunk and limbs. The efflorescences of the lids continued into the conjunctiva where the lesion was variable and changed within a few days. The differentiation from seborrhoeic eczema is often difficult.

F. Nelson.

Rössler, F. **Clearing of a band-shaped corneal opacity.** Proc. Ophth. Soc. Vienna p. 42, February 22, 1943.

A 10-year-old child suffered from iridocyclitis for the last four years and developed a band-shaped corneal opacity. Rössler removed the epithelium of the cornea and treated the area with a two-percent hydrochloric acid solution. The corneal opacity cleared up immediately.

F. Nelson.

Sauer, H. **Treatment of herpetic corneal ulcers by cauterizing with zinc sulfate.** Proc. Ophth. Soc. Vienna p. 42, Feb. 22, 1943.

Since 1925 patients with herpetic eruptions of the cornea are treated with 20-percent zinc sulfate solution which is applied with an applicator after cocaine anesthesia. Immediately afterwards the eye is rinsed with physiologic saline solution and bandaged with collargol ointment. Usually an early lesion heals within three to five days after one cauterization, older and extensive lesions within 8 to 13 days. Only two recurrences were observed. Burning pains usually persist for several hours after the treatment. It is necessary to sterilize the zinc sulfate solution by boiling because molds grow readily in it.

F. Nelson.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Alagna, Gaspare. **More about the etiogenesis of recurrent uveitis with hypopyon.** Ann. di ottal. e clin. ocul. **73**: 53-61, Jan., 1947.

A 52-year-old man had severe unilateral uveitis, in the course of which hypopyon occurred three times, and which was also accompanied by a typical dendritic keratitis. This and other cases which he has already reported support Alagna's thesis that the etiology of this very serious ocular syndrome is to be attributed to the virus of herpes simplex or at least to a herpetiform virus.

Harry K. Messenger.

Lindner, K. **Prevention of expulsive hemorrhage.** Proc. Ophth. Soc. Vienna, p. 62, Jan. 17, 1944.

In the right eye of a woman 52 years of age, iridencleisis was performed for chronic glaucoma. After the scleral incision the iris bulged forward immediately, and during incision of the iris liquified vitreous body began to pour out. The sclera was trephined at once in the temporal upper quadrant but little blood escaped. Ophthalmoscopy revealed a large choroidal protrusion in the lower nasal quadrant. Another trephining hole was put in that region after which quantities of blood escaped in intermittent pulsating spurts. After two weeks high choroidal detachment was still present but it disappeared completely within three months. Vision became 6/9 and tension remained below 30 mm. Hg.

F. Nelson.

Simonelli, Mario. **Uveo-meningeal syndrome following epidemic keratoconjunctivitis.** Giorn. ital. di oftal. 1:25-35, Jan.-Feb., 1948.

In a 32-year-old woman a uveo-meningeal syndrome appeared a few days after the onset of a typical epidemic keratoconjunctivitis. There was a neuritis as well as a perineuritis and in the absence of choroidal lesions the author attributes this to a direct transmission of the infection from the leptomeninges to the sheath of the optic nerve. All the lesions are believed to be the result of virus infection. The allergic mechanism is suggested as one method of extension.

Francis P. Guida.

9

GLAUCOMA AND OCULAR TENSION

Copps, L. A. **Cyclodialysis and iridectomy for glaucoma with cataract.** Wisconsin M. J. 47:469-471, May, 1948.

The author reviews the literature on the surgery of cataract occurring in eyes with glaucoma. His method of combined cyclodialysis through a scleral incision and iridectomy through a keratome incision at the limbus at the same operation has proved successful in five out of eight patients. These had advanced glaucoma. It seemed imperative to control the tension in one operation, a minimum of surgery was desirable, and in gonioscopic examination a large part of the chamber angle was found closed.

Orwyn H. Ellis.

Harshman, J. P. **Glaucoma associated with subluxation of the lens in several members of a family.** Am. J. Ophth. 31: 833-836, July, 1948. (1 figure.)

Jacobs, C. H. **Cataract extractions under intravenous pentothal sodium anesthesia.** Pennsylvania M. J. 51:861-865, May, 1948.

The author reviews the literature and presents a report on 77 cataract extractions done under intravenous sodium pentothal anesthesia. In the postoperative course 77 percent were satisfactory, and 10 percent had emesis.

Orwyn H. Ellis.

Sautter, H. **Subcapsular collection of fluid in a shrinking traumatic cataract.** Klin. Monatsbl. f. Augenh. 112:316-318, 1947.

Two cases of traumatic cataract after perforating injuries are reported. The cortex and nucleus underwent shrinkage, but this shrinkage was not everywhere followed by the lens capsule. The latter formed a small, optically transparent sac above the shrunken cataract. The author assumes that fluid has collected within this space, either by secretion or by invasion from the aqueous. (3 references.)

Max Hirschfelder.

Simonelli, Mario. **The effect of the administration of nicotinic acid and its amide on the manifestations of napthalene cataract.** Giorn. ital. di oftal. 1:47-52, Jan.-Feb., 1948.

Previous investigations of the author have demonstrated that nicotinic acid normally present in the lens is greatly reduced in the cataractous lens. In this paper he demonstrates conclusively that nicotinic acid or its amide, administered subcutaneously or subconjunctivally does not interfere in any way with the production of the naphthalene cataract in the rabbit, but rather tends to accelerate the rate of its appearance. This is possibly caused by the vasodilating action of the PP factor, which increases the amount of the toxin in the aqueous, and a greater stimulation of the auto-oxidative processes of the lens. Francis P. Guida.

10

CRYSTALLINE LENS

Gittler, R. **Cataracta pisciformis.** Proc. Ophth. Soc. Vienna, p. 93, Feb. 26, 1945

A rare type of bilateral cataract was seen in the eyes of a man, 45 years of age, whose father, paternal aunt, and one of his sisters likewise had cataract. The lens opacities were flat and just anterior to the adult nucleus. In addition, small, dense, ring-shaped opacities, some of them with clear centers, could be seen in front of the flat opaque layer. The zone of the senile nucleus was much closer to the lens capsule in the central than in the peripheral area. This cataract resembles that which Vogt described as pisciform and is a hereditary congenital cataract. F. Nelson.

11

RETINA AND VITREOUS

Agnello, F. **Toxic amblyopia in a subject with an idiosyncrasy to quinine.** Riv. di ottal. **2**:155-163, March-April, 1947.

The author discusses a case of toxic amaurosis, due to idiosyncrasy, in a young man who had ingested two grams of quinine. The ophthalmoscopic picture was the classic one of quinine poisoning: optic atrophy, thinning of retinal vessels and marked constriction of visual fields. The pathogenesis of the blindness is similar to that due to male fern and a result of the direct action of the drug on the sensory elements of the retina. The ischemia is secondary.

Francis P. Guida.

Appleby, R., and Pischel, D. K. **Review of 100 cases of retinal detachment.** Am. J. Ophth. **31**:796-804, July, 1948. (1 figure, 4 tables, 10 references.)

Harwood, D. H. **Solar eclipse and vision.** Ann. Western Med. and Surg. **2**: 22-223, May, 1948.

The author reports a case of macular damage that followed looking at an eclipse without proper protection. A program for prevention, especially publicity in schools, is outlined.

Orwyn H. Ellis.

Lewis, P. M. **Diathermy treatment of angioma of the retina.** Am. J. Ophth. **31**: 829-833, July, 1948. (2 figures, 10 references.)

Mallner, J. **Macular hemorrhage in spotted typhus fever.** Proc. Ophth. Soc. Vienna, p. 82, Oct. 23, 1944.

A man, 38 years of age, had spotted typhus fever with all the typical symptoms. When he awoke after 10 days of deep stupor the vision of the right eye was blurred. A small, oval, dark-red hemorrhage with the surface of a raspberry was found in the macula. Vision gradually improved during absorption of the hemorrhage to 6/18. Macular hemorrhages are extremely rare in spotted typhus fever. F. Nelson.

Tower, Paul. **Solar retinitis.** Ann. Western Med. and Surg. **2**:217-221, May, 1948.

The author reviews the literature and reports nine cases of central retinitis due to exposure during an eclipse. A plea is voiced for prevention through education of teachers and parents.

Orwyn H. Ellis.

12

OPTIC NERVE AND CHIASM

Barazzoni, Antonio. **Observations on cases of optic atrophy from methyl alcohol poisoning.** Ann. di ottal. e clin. ocul. **73**:8-27, Jan., 1947.

The present-day knowledge of methyl alcohol poisoning is summarized with especial reference to toxicology, symptomatology, ocular lesions, pathologic anatomy, pathogenesis, diagnosis, and therapy. Six cases are presented, in all of which the vision was reduced and the nerve heads showed atrophy of the simple type. Various modes of treatment were tried, but in no case was there other than temporary recovery of vision.

According to the most plausible hypothesis the toxic substances reach the op-

tic nerve by way of the central retinal vessels and are rapidly absorbed by the tissues, where they come in contact with the papillomacular bundle and cause irritation but no true inflammation. The retinal ganglion cells participate in this irritative state and in consequence undergo irreversible degenerative changes because of which all therapy is unsuccessful.

Harry K. Messenger.

Esente, Ivan. **Optic neuritis due to lactation.** Giorn ital. di oftal. 1:53-72, Jan.-Feb., 1948.

The author reviews reported cases of optic neuritis during lactation and emphasizes the differentiation between optic neuritis during lactation and optic neuritis due to lactation. In the first group the etiology does not differ from that of any other neuritis. The second group is more definitely that of a metabolic neuritis most likely due to a poor utilization or to a true deficiency of vitamin B. Cessation of nursing usually allows a reestablishment of the vitamin equilibrium in a short time. The author describes one case in which vitamin B therapy brought about some improvement in vision.

Francis P. Guida.

Klein, R., and O'Mallie, P. P. **Chiasma lesion due to fall in epileptic fit.** Brit. M. J., pp. 880-881, May 8, 1948.

A 32-year-old male epileptic fell and struck his right forehead during a seizure. Blindness and optic atrophy occurred in the right eye, and a temporal field defect in the left. No fracture was found. The vision of the right eye improved to 6/9 seven months later but this did not remain long and blindness ensued. The visual field and acuity improved to normal in the left eye. The lesion was thought to be vascular, in or near the anterior chiasmal angle. (References.)

Bennett W. Muir.

Pickard, Ransom. **The alteration in size of the normal optic disc cup.** Brit. J. Ophth. 32:355-361, June, 1948.

The author presents a large series of patients observed over a fifteen-year period. A steady and regular increase in the size of the normal cup of the optic disc was noted. The cups did not average over one-half of the size of the disc. When a cup occupies 70 percent of the disc area further tests should be made before considering it normal.

Orwyn H. Ellis.

Trávik, K. **Sudden blindness in a patient suffering from a cerebral tumor and cerebral syphilis.** Bratislavské Lekárske Listy 28:210-215, 1948.

Blindness developed in the course of a few weeks in a man, 45 years of age. The optic nerve was damaged by a tabetic process. A gummatous basal opticochiasmal arachnoiditis also existed. Glioma was diagnosed clinically, localized by arteriography and the diagnosis was confirmed at autopsy. F. H. Haessler.

13

NEURO-OPHTHALMOLOGY

Bender, M. B., and Brown, C. A. **The character of the nystagmus induced by amytal in chronic alcoholics.** Am. J. Ophth. 31:825-828, July, 1948.

Cholst, M. R. **Widening of the angioscotoma in the alarm reaction.** Am. J. Ophth. 31:821-825, July, 1948. (3 figures, 2 tables, 10 references.)

Cristini, Giuseppe. **Anomalous associated movements of lids and pupils from abnormal regeneration of oculomotor nerve fibers.** Riv. oto-neuro-oftal. 22:323-334, Sept.-Dec., 1947.

Three cases are reported showing some phenomena characterized by abnormal movements of lids and pupils due to anomalous regeneration of fibers of the third cranial nerve after trochlear paralysis. A boy of 15 years had paralysis of the

left third and fourth cranial nerves from a probable rupture of an aneurism of the circle of Willis. On looking downward or inward the left upper lid retracted upward; the left pupil at rest was larger than the right one, and it did not react in the usual tests, It became contracted on looking downward and inward. A woman, 40 years of age, after cranial trauma showed a limited movement of the right eye. At rest the left upper lid was higher than the right and on looking down it went still higher. The right pupil was dilated and rigid and the left did not react to light. A woman, 56 years of age, with persistent headaches from a probable aneurism of the circle of Willis had a right oculomotor paralysis. She exhibited the same type of upper lid and pupil anomalies as the other woman. The writer states that the sign of Fuchs is due either to an increased tonus of the lid elevator because of an anomalous regeneration of fibers to the elevator or due to transmission to this muscle of the impulses which normally go to the orbicularis. The pupillary associated movements are ascribed to an analogous regeneration of fibers. Similar phenomena are well known in neurology. They always suggest a trochlear lesion and may give a hint in the diagnosis of the site of the original lesion and the time when it occurred. (Bibliography.) Melchiore Lombardo.

Di Luca, Giuseppe. **A provoked retinal angiospasm.** Riv. oto-neuro-oftal. **22:** 309-322, Sept.-Dec., 1947.

A woman, 50 years of age, with chronic simple glaucoma showed a rapid emptying of the retinal artery when her carotid sinus was compressed by a finger. The emptying took place from the center towards the periphery of the disc. As soon as the pressure was released the empty section of the artery refilled. During the compression the patient had difficult respiration, blurred vision, tremor and paresthesia of the limbs, and a threatened collapse. The writer concludes that the compression of the carotid provoked an angiospasm of the central retinal artery with angiospasm of the small cerebral arteries, cerebral ischemia and cardiac inhibition. The carotid sinus which regulates the general blood flow has no action on the arteries of the brains. A peripheral vasomotor neuro-vegetative center must exist common to ophthalmic territories. (Bibliography.)

Melchiore Lombardo.

Joy, H. H. **Agnostic alexia without agraphia following trauma.** Am. J. Ophth. **31**:811-820, July, 1948. (3 figures, 35 references.)

Matteuci, P., and Kluzer, G. **The question of double cortical representation of the macula.** Riv. oto-neuro-oftal. **22**: 253-262, July-Aug., 1947.

A man of 33 years had short attacks of headaches of sudden onset which then became continuous. At the same time ocular disturbances consisted of a marked defect of central vision in both eyes, a right lateral homonymous hemianopsia, with a bilateral central scotoma and marked papilledema in both eyes. A diagnosis of intracranial tumor was made and trephining of the occipital region at left showed the presence of a big meningioma occupying almost the entire left occipital lobe. It is noteworthy that a tumor localized in one occipital lobe could provoke not only the homonymous lateral hemianopsia but a bilateral central scotoma instead of a simple hemianopic scotoma. Clinical and experimental data do not give clear evidence of double cortical representation of the macula. Experimental researches do not show that macular crossed fibers exist. Individual variations in the localization and also a true unilateral cortical specialization of the central vision may exist. (Bibliography.)

Melchiore Lombardo.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2
News items should reach the editor by the 12th of the month

DEATHS

Dr. Frederick William Adams, Carmel, California, died April 23, 1948, aged 88 years.

Dr. Emil Amberg, Detroit, Michigan, died April 12, 1948, aged 79 years.

Dr. William Rudisel Fringer, Rockford, Illinois, died March 7, 1948, aged 84 years.

Dr. William Elliott Gamble, Santa Monica, California, died April 23, 1948, aged 88 years.

Dr. William Burton Newton, Alpena, Michigan, died April 19, 1948, aged 67 years.

Dr. H. Lee Simpson, Detroit, Michigan, died February 3, 1948, aged 69 years.

Dr. Hiram Jason Smith, Chicago, Illinois, died February 16, 1948, aged 65 years.

MISCELLANEOUS

ENDOW OPHTHALMIC FELLOWSHIP

Alumnæ of the Upsilon chapter of the Delta Gamma sorority of Stanford University have presented the university with an endowment of $41,000 for a fellowship in ophthalmology. The gift represents proceeds of the sale of the sorority house following dissolution of Stanford sororities in 1944. Presentation of the gift, which fits into a national Delta Gamma alumnæ project for the care and prevention of blindness, followed approval by the superior court of Santa Clara County of the dissolution of the Stanford Delta Gamma association as a corporation. Sorority gifts also included a contribution to the building fund of the Palo Alto Society for the Blind.

SOCIETIES

CINCINNATI OPHTHALMOLOGICAL CLUB PROGRAM 1948-1949

The Cincinnati Ophthalmological Club announces its program for the coming year. Beginning in October, the club will meet on the second Friday evening of each month. The program will include as guest speakers: Dr. Herman M. Burian, Boston; Dr. Peter C. Kronfeld, Chicago; Dr. Alston Callahan, Birmingham; Dr. Derrick Vail, Chicago; Dr. Harold F. Scheie, Philadelphia.

Newly elected officers of the club for the year 1948-49 are: presidium, Dr. Donald J. Lyle, Dr. Ira A. Abrahamson, and Dr. Karl W. Ascher; secretary and treasurer, Dr. Joseph D. Weintraub; historian, Dr. Karl L. Stoll; program committee, Dr. K. W. Ascher, Dr. George Heidelman, Dr. Albert L. Brown, and Dr. Francis X. Siegel; committee on clinical presentations, Dr. Ira A. Abrahamson, Dr. Richard A. Hoffman, Dr. Barnet R. Sakler, and Dr. Cyril E. Schrimpf; dinner committee, Dr. Donald J. Lyle, Dr. Horace W. Reid, Dr. Victor Ray, Dr. Charles A. Hofling, and Dr. Louis J. Hendricks.

PERSONALS

HEADS CLEVELAND CLINIC

Dr. Roscoe J. Kennedy has been appointed head of the department of ophthalmology at the Cleveland Clinic. After receiving his medical degree, in 1931, at the University of Iowa College of Medicine, Dr. Kennedy interned at Harper Hospital, Detroit, and served as resident at the Buffalo (New York) General Hospital and at Wills Hospital, Philadelphia.

Dr. Kennedy came to the Cleveland Clinic in 1937 to complete a fellowship course of study and was appointed to the professional staff in 1938. During World War II, he served for four years in the medical corps of the U. S. Navy in the Southwest Pacific, holding the rank of captain.

Dr. Kennedy was certified by the American Board of Ophthalmology in 1939, and has made various contributions to medical journals. He is secretary-treasurer of the Cleveland Ophthalmological Club and is also a member of the Cleveland Academy of Medicine, American Medical Association, and American Academy of Ophthalmology and Otolaryngology. He was born at New Hampton, Iowa, and attended Notre Dame University.

WINS LITERARY PRIZE

At the exhibit of the American Physicians Literary Guild at the A.M.A. convention in Chicago, Dr. Joseph I. Pascal, New York, received first prize for his essay, "On Lecturing and Teaching."

APPOINTED PROFESSOR AT JEFFERSON

Dr. Arno Emerson Town, clinical professor of ophthalmology, New York University College of Medicine, has been appointed professor of ophthalmology at Jefferson Medical College to succeed Dr. Charles E. G. Shannon, who has retired. Dr. Town was graduated from Jefferson in 1926. He also received a Master of Medical Science degree in ophthalmology from the University of Pennsylvania School of Medicine in 1931. During the last war he served as a commander in the United States Naval Reserve as ophthalmologist at the United States Naval Medical Center, Bethesda, Maryland, and aboard the hospital ship *U. S. S. Benevolence.*

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